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**PERMETHRIN IMPREGNATED BEDNETS AND DDT RESIDUAL SPRAYING,  
MULTICENTRE COMPARATIVE TRIAL IN SOLOMON ISLANDS.**

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of Doctor of Philosophy.**

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## THE ABSTRACT

A malaria intervention comparative trial was carried out in Solomon Islands between 1987 and 1992, involving 7 pairs 14 communities, with 29,182 people. Seven communities were given bed nets impregnated with permethrin annually at  $0.5\text{g/m}^2$ , and the others sprayed biannually with DDT at dosage  $2\text{g/m}^2$ . Each pair was evaluated for about two years, by measuring entomological indices, prevalence of infection, incidence of infection, and levels of community compliance. An intensive differential cost analysis of both interventions was done in one pair of the communities and an analysis on cost and benefit of PCD mechanism was done in three pairs.

Anopheles farauti, the main vector, (A.punctulatus, an inland vector became very rare with interventions), maintained the early evening biting peak and high outdoor biting. The highest transmission potential was indoor with high parity (54.1%) and sporozoite rate (1.42%). It avoided contact on DDT sprayed surface and was not killed even though it was still sensitive with a 75.1% mortality. There were neither changes in biting density, nor parous rates with DDT spraying. Permethrin impregnated bed nets reduced biting density by an estimated 53.69%. The parous rate indoor was reduced by 11.64% when compared with that in the comparison area, and those caught in the bed nets area did not have any sporozoites. Prevalence of infection, by quarterly prevalence surveys revealed a 21.2% difference between the intervention areas after two years. The most significant decline was in the under 10 year old group ( $p<0.01$ ) in permethrin treated bed nets, including in infants ( $p<0.05$ ). The decline was especially marked with Plasmodium falciparum. There were increases in the DDT area, including

P.falciparum in the younger age groups. DDT spraying did not have any effect on the incidence of infection. Permethrin impregnated bed nets reduced malaria incidence, by an estimated 49%. This reduction was particularly significant however on children under ten years old ( $p < 0.0001$ ) and marked with P.falciparum.

Compliance with DDT declined by 30% but with bednets it remained high above 85%. These results confirmed that permethrin impregnated bednets are more effective than DDT residual spraying in controlling malaria in Solomon Islands. The operational costs for DDT spraying was \$8.53 and impregnated bed nets \$3.85 per capita per year. The mean cost of processing and examining a PCD slide is \$0.40. These cost indices took account of all materials, personnel and administration involved. It took a mean of 6.1 days from the time the smear was taken to the time examined ( $SE = 0.21$ , 95%CI 5.71 to 6.53 days). It would take twice this time for a result to be received by the health workers managing patients. Only 20% of blood slides could contribute to patient management. Based on these findings, all that is necessary is to make blood smears of patients less than 10 years of age for epidemiological evaluation of vector control interventions in malaria programme. This will save scarce resources at primary health care level. Making blood slides of everyone would not further add significant information and benefit, at an extra cost. The only exceptions are, those critically ill with malaria, complicated malaria and a patient suspected to have drug resistant malaria. Permethrin impregnated bednets are a cost effective way to control malaria in primary health care and the most cost benefit way to evaluate vector control intervention is careful monitoring of PCD results, especially with P.falciparum malaria of children under 10 years old.



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This community based comparative malaria intervention study was a very big one. It covered a large population of several communities over a wide geographical area of Solomon Islands. It involved very intensive planning, organization and management. Even though each pair was followed up for about two years, the whole research trial started in 1987, and was not completed till early 1992. It took an initial total of six months to get the trial organised after funds had been made available. It is an experience which was filled with challenges, frustrations and satisfaction, and can only be gained through undertaking such a study.

The trial involved several organisations, personnel, experts, governments and community members. It is with these joint support, co-operations and assistance that such a large study as this, was successfully completed. It was mainly the national staff, with limited essential, outside expertise that undertook the large amount of work involved. It is only justified, therefore, that they are acknowledged.

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London.

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Like all disease applied field reseach intervention studies there would be no studies without cooperating communities. The cooperation given by members of all 14 communities involved was instrumental in the successful completion of the trial. There were several problems, frustrations and happiness on the part of the field study teams, but the communities through their own kind



efforts always made the field teams happy and welcome in their communities. The amount of food provided by villagers for field teams throughout the study period would occupy several pages to list. The least the principal investigator could hope to repay this kindness is that the results of the study would further benefit these and many other communities. The modified national policy and strategies in malaria control, as a result of the study is expected to achieve just that and benefit even those who were not involved in this study.

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## CHAPTER ONE

### INTRODUCTION AND BACKGROUND INFORMATION

---

#### 1.1 MALARIA AND EFFORTS AT CONTROL

After years of intensive efforts from eradication to control, malaria is still a most important public health problem in many countries (WHO, 1990). Of the estimated total world population of 5,061 millions, 1,371 million (27%) live in traditionally malaria-free areas or where malaria has disappeared without any specific interventions, 1,617 million (32%) live in areas where malaria has been eliminated by specific antimalaria interventions and malaria-free status has been maintained. 1,599 millions (32%) live in areas where malaria was reduced or even eliminated, but where transmission is reinstated and the situation is unstable or deteriorating. This includes 1% of the world population living in areas of ecological, economic or social changes; agriculture, jungle exploitation, and sociopolitical unrest. But 474 million (9%) live in areas where endemic malaria remains basically unchanged and no national antimalaria programme was ever implemented. This occurs in the major part in Sub-Saharan Africa.

The current global statistics on malaria vary greatly with programmes concentrating on measuring infection rather than mortality. It is currently estimated (Bradley, 1991) that of the total 2,073 million people exposed to the risk of infections, 270 million are infected, 110 million are ill and one million died, mostly in Sub-Saharan Africa, where the basic health infrastructure remains rudimentary or non-existent. It is clear that with the current economic climate many malarious countries cannot sustain the level of health care people need, politicians demand, and often donor countries encourages them to adopt (Key, 1991). However there is *no quick fix recipes*



and neither is there justification for *opting out* of the immense malaria problem. As such whilst some countries still maintain the vertical antimalaria programme, others, especially in Asia, South West Pacific and South America, have attempted to integrate malaria control activities into the general health care services within primary health care. In doing so, some have attempted stratification using different epidemiological criteria and organised control activities with appropriate goals. These allow diversification and complementary measures to be applied in accordance with local conditions, resources and the level of development.

But several general obstacles have to be overcome. One is the problem with drug resistant P.falciparum. In Africa 39% are reported resistant, in Asia (including South East Asia and Pacific) 31% and in South America 13.2%. This serious problem is on the increase and includes some of the new antimalarials making case management a growing problem worldwide. New drugs that are effective and affordable by malarious countries are diminishing fast. Thus till an effective vaccine is found vector control is still the most important strategy for any hope in containing the problem. In addition the following problems (Henderson, 1991) need to be addressed and solved;

- a) insufficient financial resources,
- b) lack of knowledge about biology, ecology, and control of vectors
- c) expansion of agriculture, mining, forest industries and the development of new areas leading to migration
- d) inadequate sanitation and precarious living conditions,
- e) insufficient and non-existent infrastructures, and
- f) sociopolitical unrest.

In the last decades the increase in the number of malaria cases in many countries, arise from a growing resistance to insecticides amongst mosquito vectors as well as drug resistance



amongst malaria parasites. The conventional control of malaria vectors by spraying residual insecticide, has encountered several setbacks. These have been i) refusal of house holders to allow teams of spraymen to enter their houses, ii) increasing cost and difficulties in managing programmes, iii) resistance of mosquitoes to cheaper insecticides, like DDT, iv) organophosphate substitutes cost more, do not last long, smell worse, require expensive safety precautions, and are more toxic, and, v) tendency of some mosquitoes not to rest indoors long enough to pick up a lethal dose of insecticide.

These have been compounded by inadequate national resources, and supervision for mounting malaria control programmes with vertical administrations. These impediments are challenges to be solved in the future especially with efforts to control malaria effectively in an integrated health care services based on primary health care principles. The WHO Expert Committee, (1983), therefore advised, that a more field research approach be undertaken in order to arrive at the most appropriate vector control methods for communities in primary health care programmes.

The Solomon Islands (Fig.1) is a classical example of one such country, after years of intensive antimalaria activities, malaria is still the main public health problem causing a serious socioeconomic impact. DDT spraying which has been applied for over thirty years has now met with the difficulties described above. There is resistance by parasites to drugs, vectors to insecticide and people to residual DDT spraying. There are diminishing resources to continue DDT operations but at the same time appropriate alternatives need to be identified to provide protection to communities. Recently active efforts have been sought to integrate malaria control into primary health care. But appropriate technologies which are effective first be identified for such a primary health care approach and appropriate methods to evaluate and monitor the impact of interventions need to be determined.

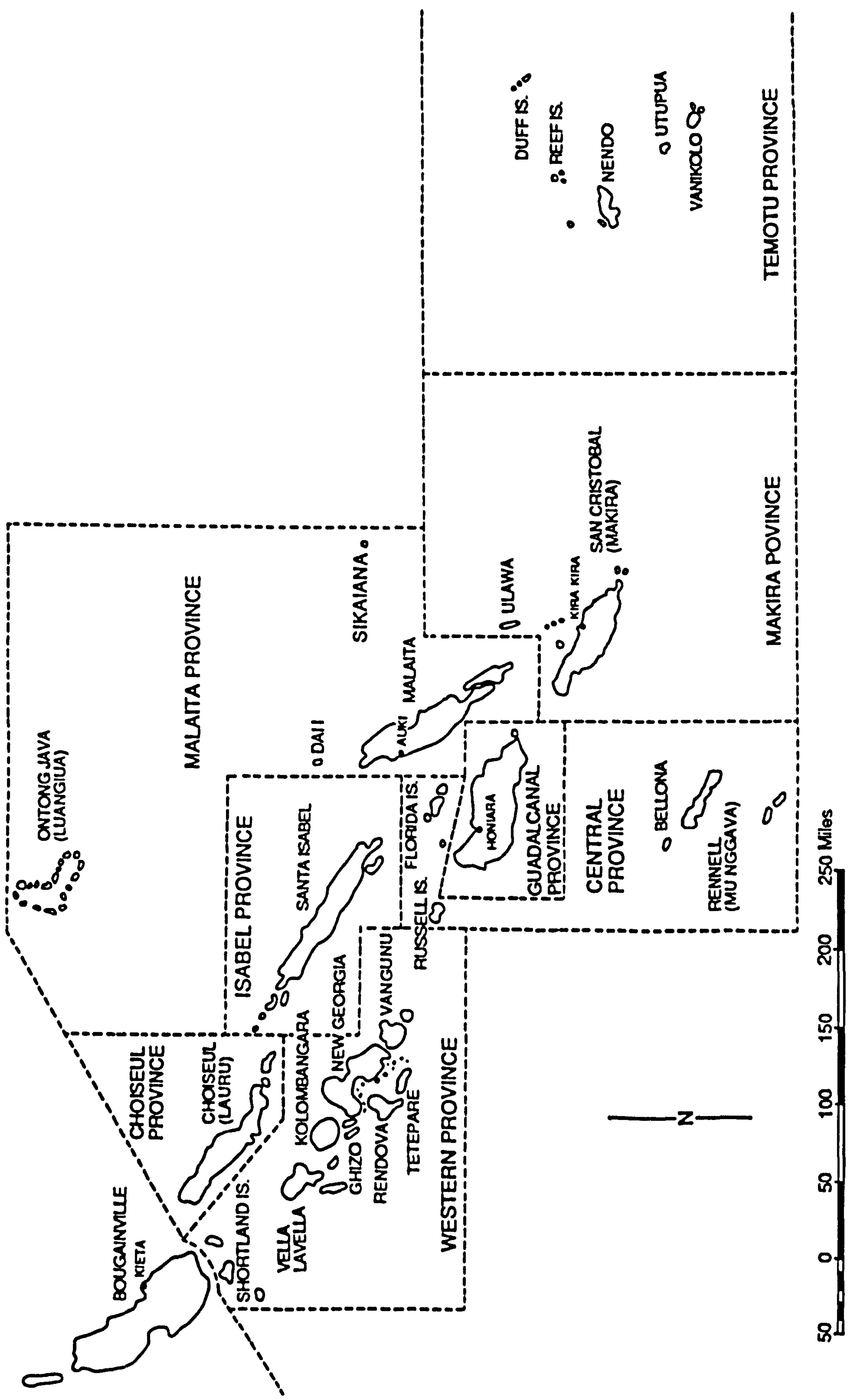


Figure 1 Map of the Solomon Islands



This is the main purpose of a large scale intervention study undertaken in Solomon Islands. The effect of permethrin impregnated bednets is compared with that of DDT residual indoor spraying. This is to determine whether the new technology, permethrin impregnated bednets, is more effective and would thus replace DDT spraying, or that it is similarly effective but costs less. It needs to be affordable and appropriate as an intervention in primary health care. In evaluating the comparative effect of these interventions, potential evaluation methods are assessed, to determine a method, or methods more appropriate in primary health care.

## **1.2 THE USE OF DDT INDOOR HOUSE SPRAYING**

### **1.2.1 Introduction**

The most widely used insecticide in public health programmes is, dichloro-diphenyl-trichloroethane (DDT), the chlorinated hydrocarbon synthesized in 1874 by a German chemist, Zeilder. Its insecticidal effect however was determined only in 1939 by Paul Muller of Geigy Laboratories, Switzerland (Pampana, 1969). This led to several successful field trials, mainly during World War II, such as that of Eddy (1947). DDT eventually revolutionized the approach to attack malaria vectors by being the first insecticide in public health used on a large scale. It is used in the same way today in some national malaria control programmes.

DDT, by reducing or eradicating malaria has improved the general health and welfare in some communities. Infant and childhood mortalities are reduced as seen in certain areas of Africa, (Bruce-Chwatt, 1984), Papua New Guinea (van Dijk and Parkinson, 1974) and Solomon Islands (Macrae, 1979). Birthweights increased significantly in Solomon Islands (Macgregor and Avery, 1975). These national eradication projects have provided useful lessons and experiences that prove most useful in developing national health service infrastructures, and programmes even in countries where malaria control or eradication have been unsuccessful (Wright, *et al* 1972). These



strategies have now enabled over 1,500 million people to live in malaria free areas, and a further 100 millions to live in areas where malaria risks have been significantly reduced, (WHO, 1986).

### 1.2.2 The Chemical and its Properties

DDT is synthesized by condensation of one molecule of chloral with two molecules of chlorobenzene in 98% sulphuric acid. The final product consists of two chlorine atoms attached to different carbons of two benzene rings giving rise to isomers. The most effective isomers are those with chlorine atoms attached to the most lateral carbons in the para-para', p,p' isomers. DDT is normally labelled 'technical grade DDT' which means that it contains at least 70% of the p,p' isomers. It is a white amorphous substance, with aromatic and slightly pungent smell. It is soluble in oil and other solvents such as kerosene (Brown, 1951) but insoluble in water. The chemical is non-volatile and has a boiling point of 107°C. It is a nerve poison, lethal to insects but irritant to certain mosquitoes which is a setback in mosquito control.

### 1.2.3 Formulation.

The most commonly used formulation is the 75% wettable powder form (w.d.p. DDT). It is formulated by mixing DDT as the active agent, with an inert carrier, often clay, by a mechanical process with wetting, suspending, and anti-caking substances added to stabilise the mixture. It is a water suspension of solid formulated DDT powder. The amount of insecticide is added until the required concentration, 75% of DDT is reached. This form has 50% to 70% DDT solid particles of sizes below 20 $\mu$  or even 10 $\mu$  which allows them to be easily picked up by mosquitoes and penetrate into their cuticles, with lethal results. It is also most suitable whilst water dries out, or penetrates into porous walls as the particles are retained on the surface (Hadaway and Barlow, 1952, 1953, Barlow and Hadaway 1958). The main disadvantage is that

particles do cause white spots on shiny surfaces. It is widely used in malaria control in the tropics where walls are made of mud (Bordas, *et al* 1953), unbaked bricks (Bertagna, 1959), thatch (Peters, 1963) and other materials.

The next widely used formulation is the 20% Emulsion Concentrate (EC). In a standard 20% DDT EC, DDT in globular particles is in the dispersed phase, and xylene, the continuous phase, with trixon-x-100 as the stabilising agent. It has also been produced with cheaper naphtha, fatty oils and ammonia. To either of these, 65 parts water need to be added before use. The advantage of 20% EC DDT is that it is less visible on sprayed surfaces than wettable powders. But the EC form is a fire hazard and requires suitable storage and is highly toxic to spraymen. Like solution the liquid will be absorbed into porous materials, making the DDT biologically useless (Hadaway and Barlow, 1953, Barlow and Hadaway, 1958). Emulsion Concentrate DDT is mainly used for spraying on well decorated surfaces, polished furniture and on shiny and non-porous walls mainly of permanent materials.

#### 1.2.4 The Actions and Doses

DDT is a nerve poison and lethal to insects that come into contact with it. It is the least toxic of the chlorinated hydrocarbons especially by the dermal route, making it safe for residual spraying (Table 1). DDT is stored in adipose tissue (Siyali, *et al* 1974) in an inactive and harmless form and is not quickly metabolised therefore persists for long periods. It is remarkable that no chronic side effects have been observed after years of continuous public health use. But DDT is toxic to aquatic and environmental life and since it persists for long periods, environmentalists have objected to its use. The only toxicity observed in humans with DDT has been acute poisoning, either accidental or suicidal. Therefore safety precautions adopted for all insecticides (WHO, 1967) also include DDT in handling, storage and application with good equipment. Even



so many of these measures have not been adopted without apparent ill effects further confirming the safety of DDT in health use.

**Table 1:**      **The toxicity levels (rats) of some residual insecticides.**

INSECTICIDES	LD <sub>50</sub> mg/kg	
	ORAL	DERMAL
DDT	118	2510
Dieldrin	46	60
Lindane (HCH)	91	900
Fenitrothion	250	350
Propoxur	86	>2400
Fenithion	254	330
Malathion	1000	>4000
Abate (temephos)	13000	>4000

**Source:** Bruce-Chwatt, (1988)

When a mosquito lands on sprayed surface it picks up DDT particles, especially on its hind legs which penetrates the cuticle and poisons it. The degree of killing is proportional to the size of DDT particles, where the optimum size is less than 10 $\mu$  (Hadaway and Barlow, 1952, 1953). The standard dosage is 2g/m<sup>2</sup> applied twice a year (WHO, 1975) and this dose was found effective with A.culicifacies in India, A.minimus in Taiwan, A.funestus and A.gambiae in Africa (Coz, 1965) and A.farauti (Peters 1963, Colbourne, 1962) in Melanesia. Its efficacy could also persist in hard, non-absorbent walls for a year. The DDT, including the w.d.p is severely damaged if stored for long periods at high temperature (Miles, *et al* 1962). It is therefore stored and transported in carbon boxes, or drums made of fibres and not in bags or metal drum to avoid destruction, especially in the tropics.



### 1.2.5 The Impact On Malaria Transmission

DDT affects malaria transmission through four main mechanisms; reducing longevity or the daily survival rate of vector, reducing vector density of endophilic mosquitoes, elimination of vectors altogether, and changing vector behaviour from being anthropophilic and endophilic to zoophilic and exophilic.

The most effective mechanism on transmission is to reduce life expectancy of the vector. This would lead to a reduction in the daily survival rate of vectors which would subsequently result in reduction of the reproduction rate (Macdonald, 1957). A reduction of survival by 0.1 through one day would reduce reproduction rate by one tenth, 0.2 by one hundredth, 0.3 by one thousandth, and so on, of the original level. If the insecticide reduces mean expectation of life ( $1/\log_e P$ ) to less than the extrinsic cycle of a malaria parasite, in days, ( $n$ ), then the mean expectation of infective life ( $P/\log_e P^n$ ) would be dramatically reduced, and even reach zero with that particular parasite. [In the model,  $P$  is the rate of survival of the vector through one day, and,  $n$ , is the extrinsic cycle of the parasite species]. When this happens malaria transmission ceases.

If mosquitoes alight on sprayed surface and pick up an adequate dose of DDT, they would be killed. This would cause selective killing of endophagic and endophilic mosquitoes. When this happens in an adequate amount the malaria reproduction rate would be minimised by a reduction of density of the vectors in relation to man ( $m$ , in the mathematical model of Macdonald, 1957). This could happen without reducing the mean expectation of life of vector populations. Theoretically, at a certain critical level of vector density relative to man ( $m$ ), transmission would cease without eliminating the vector altogether. This theory was applied in estimating the critical density of A. farauti relative to man to cease transmission of P. falciparum in Solomon Islands [Webber, 1975, Webber and Southgate, 1981]. Since malaria transmission was still continuing the critical value for malaria was not reached.

In some highly endophagic, endophilic and susceptible vectors the density will be so minimised that their oviposition will be reduced until the species itself is eliminated. This was observed with A.gambiae in Liberia, Brazil and Egypt, A.stephensi in South Iraq, A.minimus in Taiwan, and A.funestus in certain parts of Africa (Pampana, 1969). However some mosquitoes which are endophagic in some areas may not be in others. Also resistant strains of these species to DDT have re-invaded and re-established themselves (Perry, 1960).

The biting and resting behaviour of the vector may change from being anthropophilic and endophilic to zoophilic and exophilic. When this happens, and depending on the availability of animals, the vector density and mean expectation of life will remain unchanged, but malaria transmission may be interrupted. This was seen in certain parts of southern Africa where A.gambiae persisted but the human blood index (HBI) was so low that malaria transmission was practically absent, and in A.funestus with an HBI of 0.14 in DDT villages and 0.97 in unsprayed villages (Hamon, 1958). There are two possible explanations for this phenomenon. One is due to the excito-repellency of DDT, and the other is the co-existence of sibling species. When the anthropophilic and endophilic strain is eliminated the zoophilic and exophagic strains flourish as dominant, as was suspected in Swaziland (Paterson, 1963) and Zimbabwe (Davidson, 1964). Sibling species have been demonstrated in several malaria vectors such as A.gambiae (Davidson, 1958) and A.farauti (Bryan, 1973).

The criteria by which any insecticides is considered to be suitable for health use in the field, was developed quite early (Macdonald and Davidson, 1953). It should cause at least a 65% daily mortality of mosquitoes that enter sprayed houses. With at least an 85% mortality it would be suitable for the most severe malaria transmission conditions, and those less than 50% would only be suitable for moderate transmission by highly endophilic vectors. Even though the daily mortality rate with DDT varies with species and sprayed surfaces, the data indicate that the daily



mortality rate with DDT has been between 50% and 65%. Only in a few exceptional circumstances is a mortality of 85% or more observed. It would thus appear that DDT is unsuitable for hyperendemic and holoendemic areas and probably explains why in the tropics malaria has not been successfully interrupted with DDT.

#### 1.2.6 The Excito-repellent Effect.

DDT has a greater excito-repellent effect than any other chlorinated hydrocarbon (Mancera, 1960). But as insecticides differ in their excito-repellency on mosquitoes, so do mosquito species in their degree of irritability to insecticides, as was found with A.gambiae (Davidson, 1958) which was more irritated by DDT and other hydrocarbons than A.funestus. The excito-repellency of DDT could be beneficial or be a disadvantage. If the irritated mosquitoes do escape and eventually become zoophilic, malaria transmission would decline. But the most important setback is irritated vectors become completely deterred as exhibited in certain areas by A.gambiae, A.funestus and A.maculatus which escape after feeding instead of alighting on sprayed surface (Cullen and Zuluetta, 1964). In some situations the vectors wait outdoor for humans and continue transmission extradomicillary (WHO, 1987) as has been suggested with A.farauti (Taylor, 1977, Avery and Paik, 1974, Webber and Southgate, 1981).

Early studies with A.sacharovi, A.albimanus, and A.stephensi (Davidson, 1953, Hadaway and Barlow 1953, Zuluetta 1959) have demonstrated a very strong correlation between susceptibility and irritability. The irritated mosquitoes have less time to contact DDT and subsequently their daily mortality is low so at the end of 12 days, (the extrinsic period for P.falciparum), some of the original population are still alive to continue transmission.

#### 1.2.7 The Residual Activity.

Even though DDT is stable and not volatile, with time the residual deposits will lose their activity on account of scaling down, evaporation, and sorption into certain materials. The deposits can be rubbed off, covered with soot, overlaid with paper, plastered and whitewashed. These would reduce the residual activity. The residual activity with the standard dose will depend on the persistence and effectiveness of the insecticide deposit. These are respectively assessed by complicated chemical and bioassay tests which are more feasible in the field and may be all that is required. Since DDT is non-volatile and has no fumigant actions, it will lose its effectiveness if absorbed below the surface layer of pervious materials. The smaller the particles of DDT the more effective it will be, but smaller particles will be absorbed quicker into porous materials rendering it ineffective. In an early study (Hocking, 1959) particles less than  $10\mu$  took 2 to 3 days to be absorbed,  $10\mu$  to  $20\mu$  took 5 days and those  $20\mu$  to  $40\mu$  took 14 days to be absorbed into porous materials. But the larger the particles the less effective it will be against mosquitoes.

In non-porous surfaces the relationship between persistency and effectiveness is very close and assessment of one could be correlated with the other. As such the field bioassay test designed by Simmons in 1974 (Pampana, 1969) and which has since been modified is accepted as the standard WHO Bioassay test (Bruce-Chwatt, 1988). The result is read as mortality after 24 hours of exposure for both insecticide and control. Any mortality less than 10%, calculated by Abbotts correction unrelated to age of spray should be further investigated (WHO, 1960).

When the mosquito is exposed to  $LC_{100}$  it should die but if it survives it is resistant. The original interpretation of WHO test is that if 50% or more mosquitoes survive after being exposed to  $LC_{50}$  for one hour, they are resistant, but when 10% to 50% survive, there is increased tolerance. Only when less than 10% survive in the exposed would the mosquito be regarded as susceptible. Since those that survive with increased tolerance could still pass on transmission, DDT resistance is redefined (WHO, 1967) as any survival observed after one hour exposure to  $LC_{50}$



DDT unless less than 4% concentration is used. Based on these factors specific formulations are used as appropriate to the type of surfaces to provide residual effectiveness which lasts from six months to one year with the standard dose. Since DDT is quite safe the operational cost is small (Kouznetsov, 1977) compared with modern and more toxic insecticides which require expensive protective measures.

### 1.2.8 The Use in Antimalaria Programmes.

After successful trials in the late thirties and early forties, DDT was adopted in antimalaria programmes in several countries; in Venezuela (Gabaldon, 1949), New Hebrides (now Vanuatu) (Yust, 1947), and so on. These early successes initiated the concept of malaria eradication that was discussed but did not meet any support in the 4th International Congress on Tropical Medicine and Malaria in Washington in 1948 (Pampana, 1948).

Then in 1950 the 13th Pan-American Sanitary Conference adopted a resolution on malaria eradication with undefined terms but which did not change any measures that were already established in the Americas. The Expert Committee on Malaria (WHO, 1947) at that time believed that DDT was only good for recurrent malaria control. Then the very successful experiences of Greece changed all these beliefs. Malaria transmission was interrupted in 1945 in the island of Crete with DDT spraying according to all the evidences available. The evaluation in 1951 showed that malaria did not return. This provided two lessons; one is that after 3 to 5 years of DDT spraying malaria transmission was interrupted. If no indigenous case occurred within three years after the cessation of transmission, the parasite in the blood of individuals would die out (Macdonald, 1950) and DDT could be stopped in the fourth year. The second lesson is that one of the vectors A.sacharovi developed resistance to DDT after six years of use. Therefore if eradication is achieved before resistance develops, and DDT stopped, it would still be effective

should the vector return to its former level.

This led to the adoption of malaria eradication policy by the 14th Pan-American Sanitary Conference in 1954, and eventually by 8th World Health Assembly in Mexico in 1955 (Pampana, 1969). DDT was instrumental to these decision-making processes. The excitements in those early days were summarised by Black, (Black, 1974) quote,

**"The discovery of the residual property of DDT during the Second World War enabled countries to embark on malaria eradication programmes of an extent which had not previously been possible. These were so successful that it became possible to envisage the total eradication of malaria not only from entire countries but also from the whole world. There was also a warning that if this were not done quickly it might not be possible to do it at all because some malaria vectors had become resistant to DDT"**

Unfortunately throughout years of application several malaria vectors became resistant. The operational costs have also become relatively expensive and in the last decade many developing countries, where malaria is endemic, have experienced negative economic growth leaving little for health (Abel-Smith, 1986). People have refused DDT spraying in increasing numbers. It is obvious that DDT is not cost-effective enough to control malaria especially where it is highly endemic. This led to cessation of DDT spraying in several countries, contributing to a further increase in malaria cases in those countries (WHO,1987)

### **1.3 THE USE OF INSECTICIDE IMPREGNATED BEDNETS**

#### **1.3.1 Introduction.**

There is now much interest in the improvement of bednets to provide a more appropriate alternative to residual DDT spraying for malaria control. Subsequently, several investigations have been conducted on the protective effects of bednets, and the efficacy of bednets impregnated with synthetic pyrethroid insecticides. Several results from a number of early trials around the world indicate that many of the problems with residual indoor house spraying are likely to be solved by



this simple technology.

### 1.3.2 Unimpregnated Bednets

Bednets have been used for many years against man-biting insects, especially by visitors to, and inhabitants of, the tropics. If individuals maintain the nets properly, and go inside early enough before mosquitoes bite, man-vector contacts are prevented. As a consequence, transmission of mosquito-borne diseases, such as malaria could be mechanically interrupted. This concept has led to the current interest on the use of bednets in a search for appropriate and potential technology for mosquito control. Some studies (Port and Boreham 1982, Charlwood 1986), showed that unfed Anopheline mosquitoes left human dwellings where a bednet was used to search for blood meal elsewhere. The positive effect by this phenomenon was reported to have reduced splenomegaly and parasitaemia (Bradley, *et al* 1986) in children using bednets alone. But further studies showed that untreated bednets reduced sporozoite rates to a certain degree but generally parasite rates (Molineaux and Grammiccia 1980). It neither affects vector density nor circumsporozoite antibody in the community.

A setback in using unimpregnated bednets is a badly torn net which does not provide adequate protection (Curtis and Lines, 1985, and Lines, *et al* 1987). If the mesh is not fine enough to be fully insect-proof, the nets get torn in use and are not mended, the edges are not properly tucked in, mosquitoes have all night to find their way in, feed, and find their way out. Entomologists, have long known that bednets were a good place to find trapped blood fed Anophelines (Belkin, 1945). Mosquitoes may also feed through the mesh on a limb or part of the body which happens to touch the net during the night. Some mosquitoes which wait around the net are likely to bite the occupant if gets up in the night.

Therefore, bednets would protect those inside against mosquito bites, but not those outside,

even in the same room. The individuals who do not sleep inside an untreated net in the same room would be exposed to higher individual inoculation rates (Gordon and Davey, 1933) than would have been if no bednets are used and all inoculations are shared by all individuals in the room. Attempts have been made to solve these setbacks, and improve the efficacy of this technology by impregnation with suitable insecticides.

### 1.3.3 Permethrin Impregnated Bednets.

#### a. *Effect*

The early attempts in impregnation of bednets with lysol were made by government officials of the USSR in the thirties, then with DDT by both American and German forces during World War II. It was only with the advent of synthetic pyrethroids with very low mammalian toxicity, and very rapid killing on insects that really encouraging results were obtained. The pioneering studies were carried out in North America against nuisance mosquitoes by impregnation of clothing with repellent insecticides (Grothaus, *et al* 1976). The idea of impregnating fabrics by a repellent insecticide, DEET, was successfully tried in the field in Ethiopia and Kenya (Sholdts, *et al* 1976, 1977). Then came trials using permethrin (Schreck, *et al* 1978, Lindsay and McAndless, 1978) which proved most promising.

This led to increasing applied research with permethrin impregnated bednets against night biting malaria vectors. As a vector control method, and a better alternative technology to DDT in certain areas, it should at least solve the setbacks in using DDT indoor house spraying. Several studies showed the insecticidal efficacy of permethrin impregnated bednets against malaria vectors *A.gambiae* (Snow, *et al* 1987a), *A.balabacensis* (Hii, *et al* 1987) *A.arabiensis* and *A.funestus* (Curtis, *et al* 1987). Even mosquitoes found alive inside treated nets later died. Impregnation of bednets also increased the efficacy of nets with tears (Curtis, *et al* 1987, Darriet, *et al* 1984) and



also extended its protective effects (Lines, *et al* 1987) to individuals not under impregnated nets but who sleep in the same room. This phenomenon would be most beneficial if it could be effectively extended to the whole community by wide community use of impregnated bednets. Several studies tried to explain the mechanism behind this extended phenomenon. Was it diversion or direct killing that is responsible for the extended efficacy of permethrin? There were varying results between deterrence by treated nets (Darriet, *et al* 1984), or direct killing (Lines *et al* 1987, Margori, *et al* 1987). Permethrin has a very low vapour pressure (Schreck, *et al* 1978) and it is not easy to understand that there is enough vapour drifting out of houses that would be detected by mosquitoes.

Impregnated bednets also showed varying degree of inhibition of feeding and killing. Inhibition of feeding should protect persons under treated nets, whereas killing after feeding would protect the community from onward transmission of malaria as was found in China (Li Zu-zi 1986) with deltamethrin on A.sinensis and A.dirus. This community effect is the ultimate target to determine how successful permethrin-treated bednets are in controlling malaria in primary health care.

***b. Effects with different netting materials***

Studies have now shown that impregnated nylon was more effective than cotton (Hossain, *et al* 1986), even though the latter absorbed more liquids. It was also seen that absorption at the beginning of sequential dipping was the same as at the end. There was no 'stripping process' of the insecticide due to the absence of selective absorption of permethrin by all netting materials. This enabled mass impregnation by sequential dipping for the same target dosage.

**c. *Effect of washing***

Washing has been shown to reduce concentration of pyrethroids on netting materials. This was seen with automatic washing machines (Schreck, *et al* 1978) and hand washing (Snow, *et al* 1987b). The reduction in surface concentrations through washing corresponded with reductions in vector mortality except with more potent synthetic pyrethroids; deltamethrin, cypermethrin, and lambda cyhalothrin (Curtis, *et al* 1987) that are highly lethal in low doses. It is clear that until these more potent pyrethroid come into wide use, bednets would need to be re-impregnated with permethrin, every time they are washed. In China, this is done with deltamethrin annually after the New Year washing. Therefore it is necessary to have adequate knowledge of any bednet washing pattern of communities when applying permethrin impregnated bednets.

**d. *Impregnation and sociocultural beliefs.***

The generally accepted procedure for impregnating bednets with permethrin is that suggested by Schreck and Self (1985), to a final dose of 0.5g/sq.m. Since then there have been several modifications to suit local situations and needs, such as mass dipping. It is important to note that since there is no affinity between textile fibres and synthetic fabrics to cause selective extractions of permethrin, there is also the danger of losses in improper drying of wet bednets. A wet bednet that is dried by hanging will allow the emulsion to drip off the net. Therefore wet nets should be allowed to dry horizontally on the owner's bed (Lines, *et al* 1987, Snow, *et al* 1987b) till they have stopped dripping, before it is hung to dry. Insecticide which falls on the bed will result in reduction of bedbugs. This anecdotal benefit would certainly encourage communities to accept and use treated bednets. In the process of drying bednets, care should be taken to avoid direct sunlight. Even though permethrin is photostable and heat stable (Schreck, *et al* 1977), it may not be completely so when exposed to strong tropical sun in field conditions.



It is necessary to know the existing beliefs of dipping nets with those of other individuals, or in the same solution that was used in dipping the nets of others. In China bednets had to be impregnated separately as people did not like their nets to be dipped in the same emulsion containing dirt from nets of other people (Li Zu-zi, 1986). The risk with such social beliefs is that individuals might later wash their nets after impregnation, thus reducing their efficacy. It is possible that this risk is minimized by proper health education, or if impregnation is done by an aerosol spraying technique when such a technique is made available.

*e. Residual Effect*

The residual efficacy of permethrin against *Anopheles* mosquitoes have been observed to last between six months to a year in several studies (Schreck, *et al* 1978, Curtis, *et al* 1987, Kere, *et al* 1992) on most dosages adopted. Therefore bednets would need to be reimpregnated regularly, about every six months to a year depending on the dosage and community practices to maintain efficacy. The question of whether efficacy would persist much longer after several re-impregnations of the same bednet is yet to be determined by appropriate field studies. But re-impregnations with more potent pyrethroids, if their efficacy persists much longer, could be done at a longer interval, such as in China with deltamethrin.

*f. Safety.*

The low dose synthetic pyrethroid, permethrin, had been put through tests (Schreck, *et al* 1977, 1978), confirming its substantial margin of safety in humans (Leahey, 1985). Therefore it is suitable for community application in human dwellings, but is highly toxic to aquatic life. In the field, users of impregnated bednets have not encountered any side effects (MacCormack and Snow 1986) and were better off with impregnated nets than those without. The paraesthesia in

those dipping nets with deltamethrin (Li Zu-zi 1986) were not observed with permethrin. Permethrin is quite safe for human use even on direct application as a soap formulation (Yap 1986). The use of permethrin as regards safety was sanctioned by the World Health Organization, who declared that properly impregnated bednets with permethrin would pose no health hazard to those who use them. The acute oral toxicity of permethrin in aqueous solution for rats is very low, LD<sub>50</sub> being over 4000 mg/kg, while dermal toxicity is so low that it could not be demonstrated (WHO, 1986).

#### **1.3.4 Factors affecting the Use of treated bednets.**

##### **a. *Sociocultural factors***

The chemical, entomological, and epidemiological findings have all favoured the use of permethrin impregnated bednets in malaria control. But until they are accepted and properly used by individuals in communities, they may not have any significant impact on control of vector-borne diseases. There are several reasons why people would or would not use bednets including the treated ones. Evidence has shown that in communities where use is high their effect is marked (MacCormack and Snow 1985, 1986), but in communities (tribes) where ethnic reasons limit their use their effect in lowering episodes of fever associated with malaria was insignificant (Snow, *et al* 1988). Traditional and cultural beliefs and behaviours of communities, especially reasons surrounding the use of bednets, are important.

Some people, especially adults and older children, did not sleep inside bednets because they are simply 'too hot' (Hii, *et al* 1987). Such individuals run the risk of being infected outside nets. Attempts were made to solve this problem in hot sticky and humid tropics by having thinner materials with wider mesh, but wider mesh must be adequately impregnated with a suitable insecticide to make them fully insect-proof. The other possible solutions are, to use impregnated



curtains (Marjori, *et al* 1987) and impregnated eaves nets. But the latter has not been very useful during high transmission periods as it was during low transmission period (Procacci, *et al* 1991). Individuals may still use impregnated bednets but merely sleep under them with the sides rolled, which will provide a similar effect against vectors (Curtis, *et al* 1987). But these alternatives are subject to further studies in their impact on malaria transmission in communities.

**b. Nuisance value.**

Bednets are routinely used in areas where mosquito density is so high that it is perceived as a widespread nuisance. The complaints from persons, not routinely using bednets are usually they are too hot, mosquitoes do not bother their sleep, or the nets are too expensive. If mosquito density is high and the inhabitants of the house cannot sleep, they will usually find money to buy bednets, or other methods of personal protection like esbiothrin mosquito coils (Charlwood and Jolley 1984).

The nuisance factor has been adequately solved by impregnating with permethrin. Both density and human bites of mosquitoes have been reduced by wide use of permethrin treated bednets in communities (Charlwood and Graves, 1987, Snow, *et al* 1987a, Darriet, *et al* 1984, Ranque, *et al* 1984, Curtis and Lines 1985, Lines, *et al* 1987). Permethrin impregnated bednets also reduced head lice and bed bugs (Charlwood and Graves, 1987, Snow, *et al* 1987b). These would certainly improve acceptance and use of bednets including the willingness of householders to purchase the materials themselves.

**c. Cost.**

The important factor in widespread use of bednets is cost, both to individuals and governments in malaria control programmes. The bednet is the most expensive item as the cost

of permethrin which is used in low dosage is very minimal indeed. In Malaysia it was estimated that in 1985 a single size cotton net would cost US\$3.80, and nylon net US\$2.70 in wholesale price (Loong, *et al* 1986). The larger family size net would cost 50% more. Impregnating a single bednets would cost US\$0.21 each time, or US\$0.42 a year. These nets would each last three to five years and are probably within the means of most rural communities. A single bednet would therefore cost US\$1.68 to US\$3.28 for impregnation during its average life. In China a each single cotton bednet costs only US\$2.00. The cost of bednets plus deltamethrin and impregnation each year came to US\$0.065 per person (Li Zu-zi 1986). The households have been willing to purchase all the needed materials, and impregnating the nets themselves, as a result millions of bednets have been bought and impregnated. Although the bednet is an expensive item, the total cost of bednets, insecticide (permethrin), and impregnation when averaged out of each person per year, might not be beyond the reach of rural communities in malarious regions. But it is desirable that ways are found to reduce cost to local communities. Such possible ways are local production, or bulk purchase from sources that produce massive amount of nets at a reasonably low cost. If the cost of bednets are beyond the reach of local communities, careful comparative evaluations are made, especially where residual house spraying is not significantly effective. It is probable that programme funds could be more effectively used to provide treated nets to all individuals in the communities. The government could motivate communities to take part by subsidizing the cost of bednets which would promote self-reliance.

### **1.3.5 Potential use in malaria control.**

Permethrin impregnated bednets have become the alternative technology for the control of vector-borne diseases in primary health care. The impregnated nets, as well as conferring protection to individual users, are likely to kill sufficient adult mosquitoes that the vectorial



capacity of the population is reduced (Self, 1990). Subsequently, the risks of vector-borne diseases would be reduced even for people without nets.

It is likely that impregnating bednets would not only solve setbacks with DDT but be a more efficient way to use insecticides than spraying them on the walls of houses. The human odour inside an occupied net draws mosquitoes to the nets so that mosquitoes that bite indoor but exit immediately after feeding miss deposits of insecticide on the walls or ceiling but would hardly miss nets as they bite for their blood meals (Charlwood 1986). There has been some suggestions (Li Zu-zi *et al* 1986) that insecticides might persist better on netting than some types of building materials.

Permethrin impregnated bednets reduced episodes of malaria fevers seasonally transmitted by A.gambiae, and A.melas, (Snow, *et al* 1978a, 1978b). This result was likely to be due to wide use of permethrin impregnated bednets which also resulted in extensive mosquito mortality. It reduced the P.falciparum rate in children in an area of holoendemic malaria transmitted by A.balabacensis (Hii, *et al* 1987), and also by species of A.punctulatus complex (Graves, *et al* 1987). These studies confirmed that with an adequate dose of permethrin, and high rate of bednet use it is possible to reduce malaria by the use of this technology.

The largest application of insecticide-treated bednets is being carried out in hypoendemic malaria in China against malaria transmitted by A.dirus, an exophilic vector, the ricefield vectors A.sinensis and A.anthropophagus which are resistant to DDT. The previous spraying with DDT brought malaria down to hypoendemic levels, but eradication was not possible since one vector became exophilic and the other resistant (Li Zu-zi, 1986). Deltamethrin was initially used and now permethrin as well. The early result of this large application showed that even in areas of very low transmission, malaria can still be reduced even further. Since treatments were done by householders themselves, the technology has a very high potential for primary health care

programmes.

It is clear from the evidence reviewed that the use of insecticide impregnated bednets is today a most promising technology for malaria control programmes in primary health care. The limited experimental evidence available also indicates that not only is it more appropriate, but it is probably more effective than DDT. It is therefore desirable that large scale community applications be undertaken in parts of the malarious world to compare this technology with other existing technologies, like DDT. It is only by such large scale applications that the hypothesis that this new technology will control malaria, by wide-spread community use can be proven.

## **1.4 THE EVALUATION OF MALARIA INTERVENTIONS**

### **1.4.1 Introduction**

The accurate assessment of malaria transmission in man is inherently difficult, and several methods used have assumptions based on probabilities with undefined errors. Also many of the data required, such as Anopheline mosquito density relative to man, are based on the assumption that the observed values are correct determinants of the true situations in nature. There is no ideal method to measure the absolute true situation in nature. In spite of these problems and unanswered questions, relative measurements of disease transmission and the amount of disease in humans have been made and related to each other (Southgate, 1983 -Unpublished WHO mimeograph document FIL/EC/WP/83.29).

### **1.4.2 The Traditional Evaluation Tools**

The outcome traditionally used for evaluating malaria intervention has been "*parasitaemia*", expressed in rates. These are prevalence rates by surveys conducted at certain periods, or incidence rates, as annual parasite incidence calculated on positive results of blood slides of patients



presenting with fever. These variables depend on quite expensive procedures and expertise; organised surveys with teams travelling from place to place, trained staff to process and examine blood slides. The resources required are expensive.

The mechanism by which malaria staff actively search for ill people suspected with malaria is known as *Active Case Detection* (ACD). Where patients present themselves to clinics with fever, or complaints suggestive of malaria and have their blood slides taken, is termed *Passive Case Detection* (PCD). Both these detection mechanisms provide valuable epidemiological information continuously on malaria trends by communities, regions, or by age-groups, and species. The results, especially of PCD may assist health workers to treat and manage patients, and in this way may benefit patients directly.

However whilst these evaluation and monitoring tools are essential in eradication programmes, their appropriateness in a malaria control programme, in primary health care, need close scrutiny. Both methods produce a substantial amount of slides to be transported, processed and subsequently examined. These require large amount of resources; microscopists, equipments and materials, transport and communications. They, especially ACD, produce a large amount of negative slides and thus may not be a cost-benefit way of using limited available resources (Mills, 1989). Even though in theory, PCD blood slide results are to assist patient management, in rural areas where the largest proportion of malaria cases are, there is often a large interval between time blood slides are taken, sent off for examination, and results received back by health workers managing patients. It is inevitable therefore, health workers have to rely on their clinical skills in managing patients even when drug resistance is suspected. In this situation even PCD mechanism may not be cost-beneficial.

Another method related to the above is large scale episodic malariometric surveys by special teams. This method involves taking large samples of blood slides of communities at a

point in time and it provides valuable information on point prevalence, and, when repeated regularly, would also provide trend of events (incidence). Whilst this method provides valuable epidemiological information, it is quite expensive and requires intensive organization and management, and a large amount of resources. It also produces a larger number of negative blood slides. Even though regular visits may maintain good public relations in many communities, others tend to regard such visits as interfering in their normal daily activities. They see it as unnecessary when they do not feel ill. Therefore this method is an expensive way of evaluating intervention programmes and may neither be cost-effective nor cost-benefit in primary health care where resources are limited.

Examination of splenomegaly, splenometry, is another traditional evaluation method. It forms the basis for classifying malaria endemicity by the Hackett method (Pampana, 1969) which could be used to monitor changes in endemicity due to interventions. Spleen enlargement in children, ideally between 2 to 9 years, are palpated to provide an indication of whether or not they have been infected with malaria, but it has several disadvantages. It requires a well trained and experienced examiner, the results are subjective and the technique involves "intrusion" of bodies of individuals. Enlarged soft spleens of new acute cases and small grade enlargement may be missed especially in a tensed and frightened child. Whilst results depend on subjective judgement which is susceptible to bias, palpable enlargements in most cases needs repeated infections and early infections may be missed. In some communities diseases other than malaria may also cause enlargement of the spleen and this method would not be sensitive in such communities. Therefore splenometry would remain unsuitable in primary health care.

In order to determine the most appropriate method for evaluating malaria control intervention in primary health care, the specific aims and objectives of malaria control in primary health care need to be clearly defined. What does malaria control mean, reducing and maintaining



malaria prevalence, incidence of infections or clinical attacks, or mortality. This is a prerequisite so that the evaluation method adopted is appropriately sensitive to such specific objectives and aims. The objective, may, unlike eradication, not be the interruption of transmissions, thus expensive surveys and case detection mechanisms may not be justified. It may just aim at reducing exposure of individuals to malaria inoculations and cost-effective ways to evaluate such an aim need to be determined.

What is needed would be an appropriate but robust and sensitive (if possible with high specificity) tool providing maximum epidemiological information, but requiring minimum amount of resources involving adequately trained primary health care workers. Primary health care workers are mostly trained to diagnose patients clinically, take blood slides and administer treatments. This suggests the use of "clinical" rates of malaria in children caused by P.falciparum as a possible ideal for the following reasons; i) it is the most predominant malaria species, ii) children are affected by malaria more than older people as they have not yet developed adequate immunity, iii) P.falciparum has a longer sporogonic cycle, thus is more susceptible to any effective vector control interventions, iv) P.falciparum does not have the dormant stage like P.vivax, and apart from recrudescence especially with drug resistance, clinical attacks have direct relation with incidence of infections, and v) is highly sensitive to changes in inoculations, so may measure changes in malaria due to an intervention.

The disadvantages in using this variable is the effect of drug resistant P.falciparum, or partial chemotherapy. This would give rise to recrudescence out of one single infection till treated by some potent drugs, or a full course of chemotherapy is taken. However the potential for saving limited resources for only taking blood samples of children clinically suspected with malaria and only using clinical attacks by P.falciparum to evaluate intervention programmes, is a worthwhile consideration.

### 1.4.3 The Infant Parasite Rate

The parasite rate in infants with P.falciparum, is one of the most sensitive epidemiological tools in assessing malaria transmission. It is potentially a most important tool in comparing malaria control methods in primary health care, (Webber, 1992). In the early fifties Macdonald (1950) developed a mathematical model to '*measure malaria transmission*' using infant parasite rate curves to estimate inoculations. This similar concept termed '*measure of communicability*' of malaria by Moshkovskij (1967). Muench (1962) described the model as a '*catalytic process*' which is adapted to describe the disease process quantitatively.

The model was developed especially after studying the data of Earls, et al (Macdonald, 1950) with P.falciparum infection in untreated Porto Rican children, who were examined weekly for 60 weeks. He postulated mathematically that in the absence of immunity (and chemotherapy), if mortality is negligible, and transmission is stable at an equilibrium, the probability that one single inoculum leads to an infection (affected) and the probability for it to revert to the uninfected (unaffected) state, in one day, are the same. This indicates that an inoculation would give rise to an infected state, which subsequently leads to an uninfected state, per unit time.

The mathematical formula is as follows;

$$L = \frac{h}{r} \dots \dots \dots (1)$$

- where,
- $L$ , is the proportion of infants infected (limit) when transmission is stable, and undisturbed, therefore at unity,
  - $h$ , is the proportion of the population receiving infective inocula in unit of time (inoculation rate), and,
  - $r$ , is the proportion of affected people who have received one infective inoculum only, and who revert to the unaffected state in unit of time (recovery rate).

However at any point in time,  $t$ , the proportion of people affected (infected),  $x$ , is

determined, with  $e$ , the base of natural logarithm, by the formula;

$$x(t) = L[1 - e^{-rt}] \dots\dots\dots(2)$$

But when transmission is in a state of equilibrium,  $L = 1$ , and  $h = r$ , then the above equation can be simplified to;

$$x = 1 - e^{-h(t)} \dots\dots\dots(3)$$

This curve plotted by the mathematical model of Macdonald fitted well with early data from Kissi, Nigeria (Davey and Gordon 1933), and from Sierra Leone (Walton 1947). Therefore at the point where transmission is at an '*equilibrium*' the maximum proportion of infected that can possibly be reached is unity (1 or 100%). But in situations where the recovery rate is greater than the inoculation rate the proportion of the population affected (prevalence rate) at a point in time is still determined by equation (2). In applying the above concept, the probability of an infant becoming infected increases with age, with increased exposure to inoculations (Davey and Gordon, 1933).

In the situation where transmission is stable at equilibrium (and reaches unity), daily inoculation rate can be parasitologically derived by modifying formula (1) as below;

$$h = rL \dots\dots\dots(4)$$

The one problem with equation (4) is that in certain situations the inoculation rate may be larger than recovery rate, but the proportion infected can only theoretically be stabilised at unity. In such situations the inoculation rate estimated by this equation, would be an under estimate of the true value.



In his calculation Macdonald (1950) derived the value 0.005 for daily recovery rate of P.falciparum, or that an infected person has an 0.005 chance to become unaffected per day. The concept was applied in epidemiological investigation in the field by Pull and Grab (1974), Dietz, *et al* (1974) and also by Mahoney (1979) in his epidemiological study of bovine babesiosis. The theory in this concept is that under conditions of stable malaria, it can be assumed over the time interval considered, that the infant population is exposed to a constant force of infection. This is measured by the number of infective contacts (inoculation rate) per susceptible child per day, which is evidenced by blood smears positive for malaria parasites.

#### 1.4.4 Field Application

The model was tested by Pull and Grab (1974) who studied cohorts of infants in Kenya. They followed up the cohorts parasitologically and also collected corresponding parasitological data. In using the entomological data, the proportion infected was theoretically calculated, and was found to correlate well with the observed proportion of infants infected with the same species. Similar studies also took place in the Garki project (Molineaux and Gramiccia, 1980) again exploring the usefulness of infant parasite rate in assessing malaria transmission.

Inoculation is the most important variable in this model which measures the association between it and parasite incidence and prevalence. Some early studies indicated that only the infant parasite rate of P.falciparum would epidemiologically provide a meaningful association (Davey and Gordon 1933) in the presence of control measures. But unless carefully designed data collection and analysis is made (Walton 1947, 1949), any observed association, or lack of association, could lead to serious errors in comparing control methods. This is so because the inoculation rate varies with season and location, as well as individual inoculation risks (Davey and Gordon 1933). When more people are exposed and equally share the same number of inoculations,

the individual inoculation rate is reduced. This would consequently affect the direct association with the proportion infected.

#### 1.4.5 Selective feeding of mosquitoes in infants

The difference in individual inoculation rate is also influenced by selective feeding of *Anopheles* mosquitoes on individuals (Davidson and Draper, 1953, Pull and Grab, 1974, and Dye and Hasibader, 1986). Therefore such selection affect the parasite prevalence and incidence in different individuals or age-groups. The vector *A.albimanus* appeared to have preferred to bite adult male Jamaicans and least the youngest female children (Muirhead-Thompson 1951). In large African families *A.gambiae* preferred adult males, while in small families the differences were not as obvious (Thomas 1951). Since then, several studies have shown other factors which have an impact on the feeding preference of *Anopheles* mosquitoes; ABO blood groups (Bryan, *et al* 1978), age-groups (Clyde and Shute, 1958), mother versus infants (Spencer, 1967 and Boreham, *et al* 1978) and others (Boreham, *et al* 1979).

Recently it was shown that *A.gambiae* preferred to feed on larger and heavier adults with larger surface area and body weights (Port, *et al* 1980). Infants only received a proportion of inoculations received by adults, determined entomologically, and this proportion should be used as a correcting factor to estimate inoculations received by infants. For example according to Macdonald (1957) the inoculation rate could be determined by the following formula;

$$h = mabs \dots\dots\dots(5)$$

where, *ma*, is the man-biting rate,  
*b*, the proportion of mosquitoes with sporozoites that are actually infective,  
 and,  
*s*, the proportion of mosquitoes with sporozoites in their salivary glands.

The inoculation per infant should be derived by correcting equation (5) by a factor,  $f$ , which is calculated below;

$$f = \frac{\text{proportion of feeds on infants}}{1 - \text{proportion of feeds on infants}}$$

Therefore the entomological inoculation rate in infants with modification of equation (5) is as follows;

$$h = mabsf \dots\dots\dots(6)$$

In a study with A.gambiae, (Port, *et al* 1980) a correcting factor,  $f$ , of 0.28 was derived. This means every time an adult receives one inoculum, the infant has an equal chance to receive 0.28 inoculum, or a 0.28 chance to receive one inoculum. This needs to be borne in mind when estimating inoculation rate by entomological methods and comparing it with that derived by infant parasite rates. As seen with A.punctulatus complex, the inoculation rate also varied between locations (Saad, *et al* 1980, Charlwood 1985). Ideally both entomological and parasitological data should be collected from the same area. The proportion infected would only reflect the impact of the intervention on the vector population of that specific area. It should not be extrapolated to others where entomological situations may be totally different (Molineaux, *et al* 1978, Pull and Grab, 1974, and Payne, *et al* 1976).

#### 1.4.6 Efficiency of Sporogony by Vectors

The dynamics of infective inoculation risks in an area and efficiency of sporozoite productions by vectors are related. The sporozoite rate is a strong determinant of the inoculation rate as are formulae to estimate inoculations (5) and (6). It has also been shown that the



inoculation rate which depends on sporozoite rate, is different with different malaria species. A recent study with species of the A.punctulatus complex (Burkot, *et al* 1978), explained one of the reasons why P.falciparum is transmitted more effectively than P.vivax. This is due to the high efficiency in producing larger number of P.falciparum sporozoites than P.vivax, per oocyst. It was demonstrated that per oocyst there was a geometric mean of 2,240 sporozoites of P.falciparum, but only 200 sporozoites of P.vivax. Also on average, per mosquito, there were 4000 sporozoites of P.falciparum, and only 380 sporozoites of P.vivax. Thus in a situation with high inoculation rate, transmission of P.falciparum would be much higher and efficient than P.vivax. Changes in P.falciparum is therefore more highly susceptible as consequence of effective vector control methods. A reduction in the longevity of the vector would have more impact on the inoculation rate than mere reduction in vector density. Therefore in this regard P.falciparum rate is an ideal variable to be adopted for evaluating vector control measures.

#### 1.4.7 Incubation and intrinsic periods of parasites.

There are two important factors regarding parasite species that may make it suitable as an assessment tool for malaria transmission; incubation period and the intrinsic period (asexual stage) inside the human host. In order that a species be ideal, both periods of that species should not be interfered with by confounding variables which will make estimation of the time of exposure to inoculations unreliable. In 1980 the post-sporozoite hypnozoite form of P.vivax in hepatic tissue was demonstrated and described (Krotwoski 1985). This dormant form is responsible for 'relapses' when it is reactivated at a later period which may vary from weeks to years. Since there is yet lack of knowledge to determine this period accurately, this species is epidemiologically not sensitive to use with the onset of disease to estimate the time of exposure to inoculation.

The next important question therefore is the incubation period of P.falciparum malaria. In

order to pin-point the time of exposure, the shorter the incubation period the easier is the determination of the time of exposure. Under ideal condition, the average incubation period of this species is 10 days. Therefore in infants, especially under one month old, the estimated time of exposure should be corrected by subtracting 10 days. Any P.falciparum infection in infants less than 10 days old, assuming that infection from prevailing mosquito inoculation is only possible at one day old, would be congenitally acquired. Congenital P.falciparum malaria, like any congenital malaria is epidemiologically not a sensitive measure of malaria transmission by direct inoculation.

#### 1.4.8 The Gametocyte rate in community.

The host infective pool is an important factor in transmission. Several early studies (Barber and Olinger 1931, Robertson 1945, Macdonald 1952), failed to agree on the concept of minimal threshold of gametocyte density that would infect *Anopheles* mosquitoes. There are several factors affecting host infective pool that are yet not fully understood; the differences in malaria species, effects of immunity (and chemotherapy), which gametocytes are infective, the chances that both sexes of the same species are ingested by the same mosquito to propagate the sexual stage of sporogony, and so on. Therefore it may be practically adequate to simply adopt the index, *gametocyte rate* in a population rather than density of gametocytaemia to predict the chances of infecting mosquitoes to continue the development of the parasite to sporozoites which will be inoculated into human hosts. The presence of malaria in humans is measured as *parasite rates*. In a study with P.falciparum in Papua New Guinea (Cattani, *et al* 1986) there is direct positive correlation between observed parasite incidence rates and gametocyte rates, by time and location. It was demonstrated that gametocyte rate and density decreased more rapidly with age, than parasite rate and sporozoite density (Dietz, *et al* 1974, Cattani, *et al* 1986). This suggests that

immunity reduces infectivity before increasing recovery. Any reduction to P.falciparum prevalence would not only itself be a reflection of effective control, but further reduce transmission by reducing the infective pool for mosquito vectors. The gametocyte rate is highest in young children who thus form the infective pool in the communities.

#### 1.4.9 The Effect of immunity

Immunity as an important confounder to the infant parasite rates, has long been considered (Macdonald, 1950). In an area of continuous transmission, when chemotherapy and other control measures are disregarded, the older the infants the higher the immunity develops and subsequently the higher the recovery rate. The parasite rate would therefore drop in the older children. It was observed in the Garki Project (Molineaux, *et al* 1978) that the level of P.falciparum antibody titre in unprotected infants fell to a minimum at 25 weeks, then increased, and by between 350 days and 700 days the proportion of immune infants was close to the cumulative prevalence of the parasitological conversion rate. In infants born from protected populations, the level of P.falciparum antibodies started at a lower level than the unprotected, but fell to low levels and after 25 weeks remained lower and may even decrease further. These results, though would depend on the sensitivity and specificity of the test and showed how important an impact on immunity would affect parasite incidence in specific age groups, even with interventions.

During the transmission period with seasonal malaria in Kano State, Northern Nigeria (Diez, *et al* 1974), the prevalence rate of P.falciparum increased to the highest by age-group 1 to 4 years. After that it fell with increasing age as a result of increasing immunity, expressing itself as increasing recovery rate or decreasing susceptibility. In areas of stable malaria transmission with a steady challenge of the antigen, immunity may develop earlier, except in situations where interventions effectively protect infants and young children from exposure to inoculation.



This review indicates that it would seem justifiable to simply use the parasite rates with P.falciparum in infant, or young children, to evaluate vector control methods. A method that is effective will minimise transmission so low that the parasite rate will not increase up to the level observed in the unprotected (or protected by a less effective method) at the same age. Thus comparing incidence and prevalence data on specific age-groups of children in areas of interventions is all that is probably required. The observed parasitological data on infants or young children with P.falciparum is therefore a potential tool to assess malaria transmission by different vector control methods.

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## **CHAPTER TWO**

### **MATERIALS AND METHODS**

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#### **2.1 JUSTIFICATION AND PURPOSES**

In efforts to control malaria as an integrated approach within health care services, effective and appropriate interventions in primary health care need to be clearly defined. This is particularly so when traditional interventions, such as DDT, (which may have not been entirely effective, but continue to be implemented), need to be replaced. When the best intervention is determined it is desirable that affordable, feasible and appropriate, but sensitive methods to evaluate the intervention are also established.

This large scale community based field study has the following purposes;

- a. To compare DDT residual house spraying with permethrin impregnated bednets, and,
- b. develop a simple, cheap, but sensitive method of evaluating malaria interventions in primary health care.

A community based trial such as this also allows evaluation of other factors, including managerial and operational which are important in primary health care consideration. This is the justification of this study, the result of which may not only benefit the Solomon Islands to control malaria, but provide equally valuable lessons to other countries with similar malaria problems.

#### **2.2 THE SPECIFIC OBJECTIVES**

The followings are the specific objectives of this large scale community-based field trial;

- a. To set up, implement, and evaluate a comparative field trial between DDT house spraying and permethrin-treated bednets, with a study power of 90% to detect a 50% protective effect of permethrin-treated bednets over DDT house spraying at 5% significant level, and,
- b. assess the relevance of P.falciparum malaria in children under 10 years, collected by prevalence surveys and the PCD mechanism, in measuring the same differences between the two interventions, at 5% significant level.

### 2.3 SUBSIDIARY OBJECTIVES.

The study provides excellent opportunities to obtain other useful information. Many of these are required to complement any decisions that the main objectives, if achieved, are appropriate in primary health care. The subsidiary objectives therefore are as follows;

- a. To assess the impact the two interventions have on the transmission potentials of the local vectors of malaria; Anopheles farauti and A.punctulatus, on their density, parity, and sporozoite rates,
- b. assess community understanding and compliance to both interventions,
- c. undertake an economic assessment of both interventions in terms of operational costs of each programme per person served, and
- d. assess the cost, benefit and usefulness of PCD mechanism the current principal method of evaluating and monitoring control programmes.

### 2.4 THE STUDY POPULATION

#### 2.4.1 The Unit of Study

The unit of study in this large comparative field trial is the community. The objectives of



the interventions made it desirable that this was the most logical design within the prevailing local situation of available resources and funds. Both interventions were applied throughout the selected communities. It was easier and more acceptable politically and ethically to apply them throughout a community. It was also more acceptable according to local requirements to have a community of villages randomised, than villages within communities, or households within villages.

#### 2.4.2 Study Population Selection

The number of communities in each of the pair, one for permethrin treated bednets and the other for DDT, were determined using the comparison of means, with the following formula;

$$n = [(z_1 + z_2)^2 (\sigma_1^2 + \sigma_2^2)] / (\mu_1 - \mu_2)^2$$

where $z_1$ ,	denotes the significance level,
$z_2$ ,	the power of study,
$n$ ,	the number of study communities in each group,
$\sigma_1$ ,	
and $\sigma_2$	are standard deviations of the group with intervention (permethrin treated bednets) and other group as 'control' (DDT) respectively, and
$\mu_1$ ,	
and $\mu_2$	the respective means of the groups.

This calculation was based on the assumptions that the standard deviation detected in the pre-intervention study, then assumed equal, would not change in both groups, and the mean would not change in the DDT area, while that of permethrin treated bednets area would have a 50% reduction. The following have been predetermined; the study with 90% power to detect a 50% reduction in the permethrin areas at 5% significance level.

Pre-intervention surveys which were conducted in some selected areas revealed a mean prevalence rate of 27% in the under 10 years old, with standard deviation of 5.3. Based on these information and using the formula for comparison of means the required communities in each of

the two groups were calculated;

$$n = [(1.96 + 1.64)^2 (5.3^2 + 5.3^2)] / (13.5 - 27)^2$$

$$= 4 \text{ communities}$$

However in study of transmission of infectious diseases there are invariably variations within and between populations, month by month, and even year by year as related to climatic, demographic, vectorial, socioeconomic, and as well as other factors. These heterogenic factors would give rise to variations in standard deviations in both groups, as well as the mean in the DDT group. Therefore 7 pairs of communities were studied in each group instead of the 4 pairs calculated. This was the maximum size that was operationally feasible with available funds and resources.

After the number of communities were selected, the size of the sample within each group was calculated using the same pre-intervention data and that the sample was adequate at 95% precision to detect the same level of significance if there was a difference. This was calculated as follows. The standard error of the samples were calculated using the following formula;

$$SE = \sqrt{[(s_1^2/n_1) + (s_2^2/n_2)]}$$

Using the same pre-intervention standard deviation for each group and pre-intervention samples of 563 in DDT and 675 in bednets, the standard error (SE) came to 0.19. It was intended that the sample should have a 95% precision, thus the size of the standard error required was,  $e = 1.96 \times SE = 0.37$ . The sample required for each group was calculated using the formulae for comparison of means;

$$n \geq (\sigma_1^2 + \sigma_2^2)/e^2$$

This came to 410 for each group. But allowing for non-compliance, heterogenic factors and other

sociopolitical reasons, the samples were increased. Using the above as number required the actual number per group was determined by weighted average of the population in the two groups. The villages were again selected at random till the number required per group was obtained. The final sample size came to 2100 for DDT and 1473 for permethrin treated bednets, increasing the power of the study. These villages were then followed up by quarterly prevalence surveys during the study period for about two years.

### 2.4.3 Allocation of Interventions

The standard scientifically accepted design to assess disease interventions is by randomized '*blind*' or '*double-blind*' case-control study (Smith, 1987). The allocation of interventions are in such a way that the participant (or assessor) is not aware of who is receiving the intervention in a '*blind*' (or single blind) study. When both assessor and participant (or participating community) are not aware of who is receiving the intervention, the study is '*double blind*'. These are necessary to minimise or avoid biases in responses, and handling and processing of data.

It was not possible to carry out a *blind* or *double-blind* trial because of the considerable cost of supplying and impregnating bednets with a placebo, or spraying the walls with an insecticide-like substance. Whilst this is so, efforts were made in the design and implementation to ensure a rigorously scientific research study involving human beings was as feasible and ethical as possible. Consequently, pairs of communities with as similar underlying pre-intervention conditions as possible were 'blocked' off (Smith and Morrow, 1991) for allocation [Fig.3].

This '*block*' technique took into account the following factors. Each pair of communities were as close as possible with similar weather and environmental conditions, but divided by sea, or uninhabited zones (buffer) of at least one kilometre between the bordering villages. The communities have been known to have a high compliance with DDT spraying, and high



endemicity of malaria. There was cooperation by the communities, authorities and government existed, and they were accessible. Migration was estimated to be less than 10% (National Census, 1986), to minimise 'contaminations' between areas.

A too strict control of the study communities was not intended so as to allow some opportunity for 'normal' non-compliance which would provide realistic outcomes as consequence of the interventions. But committed efforts in active health education were consolidated to maintain as high a degree of compliance as possible. Which of the pair received intervention was randomly selected using random numbers [Table of Fishers & Yates (1963)]. The community that was allocated with an odd number remained as DDT area and the community with an even number was allocated permethrin-treated bednets.

## 2.5 THE OUTCOME MEASURES

### 2.5.1 Introduction

Outcomes, or endpoints, are important aspects of any disease intervention study and an outcome may be termed '*a case*'. It is a prerequisite that outcomes are clearly defined and when cases are used, what constitutes a case is also defined. It is essential that the selection of the outcomes is relevant and appropriate to the objectives of the intervention study. In this case the determination of an effective malaria intervention, and, an evaluation of appropriate tools in primary health care. The following outcomes were studied.

### 2.5.2 The Prevalence of malaria infection.

For this study prevalence was defined as the *proportion in the sample that have malaria parasites detected in their blood smears at a point in time*. This point in time was the time of the prevalence survey.

Prevalence surveys or mass blood surveys of the selected villages in both groups (C in Fig.2) were done at quarterly intervals. Surveys were done once or twice before intervention and repeated quarterly to provide a trend of point prevalence. Mass blood surveys were well accepted by communities as they had been the main components of malariometric evaluations since the early antimalaria activities. Blood smears, each with a thick and thin film of all individuals, in selected villages, were taken by a team of field technicians. They were initially examined microscopically under Giemsa stain either in field laboratory or provincial laboratory. The thick film was examined for parasites and the thin smear for species and where possible density, parasites per 300 leucocytes. A negative smear was declared after examining 100 fields of thick film under high power without detecting a parasite. This initial examination in the field or provincial laboratory allowed those found positive with parasites to be treated by the field staff to maintain effective public relations in the communities.

The blood slides were then sent to the parasitology laboratory, Medical Training & Research Institute, Honiara, clearly identified except the results of field examinations. They were re-examined by the microscopists who mostly had not visited the field as a routine. Both results were compared and any differences were cross checked and errors corrected. In this manner the control of quality was maintained and since the laboratory examination was objective, its sensitivity and specificity were estimated to be high and results reproducible. The cross checking minimized any examiner errors (bias). There was no problem with colour blindness as anyone found to be colour blind was not trained as a microscopist. The observer errors due to fatigue, boredom, and so on were minimised by careful programming of activity, as well as cross checking. It was therefore expected that the result of these prevalence surveys would be best estimates of true prevalence of malaria infection as a consequence of the interventions.

### 2.5.3 The Incidence of malaria infection.

This outcome is based on certain factors. One is that individuals who become ill with malaria such as children, individuals debilitated with other illnesses and individuals who have contacted malaria are radically cleared of antigens and do not develop immunity by antigen-antibody reaction. They are thus susceptible to malaria and when they are infected by a mosquito they develop malaria illnesses. They therefore provide a sensitive measure of incidence of malaria infection.

For this study Incidence was defined as *the proportion of new cases of microscopically proven malaria illnesses occurring in the study populations within a given period of time*. This is illustrated (B), in Fig.2. This period of time was monthly each year. For this variable three pairs of study communities were again randomly selected. The reported positive PCD blood slides from the PCD agents in these communities were carefully monitored. The small proportion that would be missed were those that did not seek health care and also those that attended health care when there were no clean glass slides and lancets available. As discussed elsewhere, studies have shown that with extensive primary health care coverage only a few did not seek health care. The latter were minimised by making absolutely sure that supplies of clean slides and lancets were always available. Much of this had been achieved by the general services, but they were re-emphasized in the study areas.

The blood smears made from feverish or ill individuals were processed and examined in the same manner, except that the PCD mechanism was an on going process. The results were sent back to the originating health worker who managed the patient. Each laboratory reported weekly returns on all positive cases to provincial and national levels. Any late returns were chased up actively. This measure, being mostly objective laboratory tests, was also expected to have an adequately high sensitivity and specificity, and may be reproduced by laboratory microscopists.



However the quality control of the process was by strict cross checking of all positive blood slides and 5% of negative slides, a system which had been set up by the malaria control programme. In this way the accuracy of microscopists were maintained and assistance offered when required.

The PCD agents and laboratories serving selected communities were visited at the outset and were informed of the objectives and advised only to use the same name and complete particulars of each individual presented to them for health care. In the processing of data all individuals were coded by their names, ages, sex and village. A sample of these were checked and were found to be correct. This would enable analysis later to exclude those same individuals appearing as positive with the same species within four weeks. These would be either recrudescence due to impartial treatment or resistance, and not new infection as in the above definition. This outcome in the final analysis mainly concentrated on P.falciparum malaria which was the main species of interest. The analysis of such data especially in children would provide a more sensitive picture of malaria transmission in relation to interventions.

#### 2.5.4 Other outcomes

##### a. Impact of Interventions on Malaria Vectors

The collection of the following outcomes included both principal vectors in Solomon Islands A.farauti and A.punctulatus. The data were collected in normal residential shelters in villages, and also by experimental huts, specially designed and erected in each of the intervention areas. This included an area without intervention, for more intensive entomological studies. The entomological outcomes measured as indicated in the conceptual framework, (D) in Fig.2, were the followings.

##### i) The changes in biting density.

The man-biting rate, which was defined as *the number of female Anopheline mosquitoes*

*biting per person per hour* was monitored in the DDT and permethrin areas, as well as control. Data were collected both indoor and outdoor. The technician using a plastic sucking tube caught mosquitoes that landed to feed on them. This was done initially prior to intervention (permethrin) and during the same period twice a week for 18 months. The mosquitoes collected were counted and dissected for parity the next day. The thoraces were preserved and sent to the Queensland Institute for Medical Research (QIMR), Brisbane, Australia for sporozoite determination by ELISA.

ii) **Changes in the biting behaviour.**

This information was collected by technicians catching landing female Anophelines throughout the night both indoors and outdoors. It was done in all areas once a month due to limited number of technicians for entomological data collection. The collected mosquitoes were calculated as landing density (man-biting rate) per each hour throughout the night.

iii) **Changes in the infectivity potential of the vectors.**

These were determined by parity and sporozoite rates. Parity was measured as parous rate which was defined as '*the proportion of female mosquitoes that had laid at least one batch of eggs*'. This general rate however was not highly sensitive to accurately determine infectivity of vector populations even though it provided a most useful indication. A mosquito, which fed every other day, needs a blood meal to fertilize each batch of eggs and could only be infected with the first blood meal. It would need at least five blood meals to become infective for P.vivax, or 7 blood meals for P.falciparum, in ideal condition. But once infective a vector would continue to be so till it dies. Therefore the parous rate, which was feasible to measure with existing technology gave some indication of potential infectivity of the vectors.

The sporozoite rate, which was defined as '*the proportion of mosquitoes sampled that have sporozoites*' is the most sensitive measure of infectivity. Samples of mosquitoes collected were preserved and sent for examination by ELISA technique to the Queensland Institute of Medical Research, Brisbane. This technology has now been established in the national Medical Training & Research Institute, Honiara, Solomon Islands. The sporozoite rates in the intervention areas were analyzed for the relative difference in infectivity of their vector population.

**b. Community Compliance to intervention**

The outcome, Community Compliance, was defined as *the proportion of population accepting and using the interventions*. Eight villages, each for DDT and bednets groups, were randomly selected. In these villages a questionnaire/observation survey was conducted using a structured format (Annex A). This was in the form of a KAP study. The surveys were done prior to introduction of bednets and repeated 14 months later and a small selected survey 20 months later. In each selected village, the head of the household was interviewed using the pretested structured questionnaire. Then during the evening between 1900 hours to 2200 hours surprise visits were made to note how many members were in the house, and how many were under the bednets. The shelters in DDT areas were inspected for evidence of recent DDT spraying.

The first questionnaire/observation survey was done by eight specially selected and trained interviewers who also did the pretesting. The second survey was done by all except two previous interviewers and the third one by only four of the ten original interviewers.

**c. The Economic Impact of Interventions**

This study provided several opportunities for assessing other outcomes such as economic impact of interventions, either direct or indirect (Shepard, 1991). But several of these needed more



staff, resources and time. However since some economic outcomes were desirable for decision making processes when results of this intervention study were considered, two were measured as conceptually represented in (D) in Fig.2.

#### **i) Cost of Interventions**

The operational costs of DDT residual house spraying and permethrin treated bednets. The geographical "mid-zone" of the study communities, Ngella Islands, was selected for this assessment mainly based on logistic considerations. Data were collected since 1988 when DDT spraying was carried out in the community. The following year when the comparative trial started, permethrin was distributed to the same community. During two years the following data of the two interventions, were collected. Total capital costs of the compounds and equipments used, the total cost of transportation right down to application sites in villages in terms of fuel, oil and transport equipment, salaries and wages of all those involved in the total time of operations, and several other administrative and management costs. This gave the direct operation cost of the interventions.

All capital equipment such as transport equipment, sprayers, impregnation equipment and so on that were reusable, including bednets were annualized for cost per useful life. The cost in proportion to their being used in the operation were subsequently calculated. The costs were later adjusted to the 1990 cost and analysis made to derive the annual comparative cost of operation per capita of the two interventions.

#### **ii) Cost of PCD Mechanism.**

The cost of glass slides, Giemsa stain, alcohol for fixing thin slides, buffer tablets, anosol (oil) for reading slides and other stationeries such as wool, papers, forms and so on were obtained.

Then the average microscopists' time were calculated and the mean number of glass slides examined per day. The average salary of microscopists were also obtained. Finally the cost of microscopes was annualized per year of active life. These variables were used to calculate the average cost of examining a blood slide for malaria parasites.

This cost however did not take account of transportation, laboratory space cost, water, electricity, nurses' and other PCD agents' time in making smears. Then randomly selected positive cases from the study areas were obtained by weighted average to provide a suitable sample representative of all slides taken in those areas. The time interval between blood smear being taken to it being examined microscopically was calculated for all selected cases. The mean time taken for a blood smear to be examined, was calculated. Calculation was also done excluding slides which were taken at health posts where laboratories were situated. Assuming that the result of the blood slide took the same mean time to reach the originating health worker the mean total time for a blood smear to be taken and the result received was calculated. These calculations were used to determine the benefit of PCD mechanism.

## 2.6 DISCUSSION ON OUTCOMES.

The outcomes discussed above are represented in the conceptual framework in Fig.2. When an intervention is being applied to a community it may only be proven effective if there is a high community compliance. *Compliance* as an outcome (A) was measured, but suppose compliance is lower than required and a person either becomes sick with malaria or not. Let it be assumed that compliance is lower than the minimal threshold required to avoid inoculation and thus infection, several persons in the community would become sick with malaria.

A sick person, depending on available services, has a choice whether to seek health care or not. It has been observed that with the prevailing situation in the Solomon Islands, the majority

with malaria illness (Briese *et al*, 1989, Fardy *et al* 1990, Kere and Quan 1992) seek health care. The few who do not seek health care, either take no treatment, treat themselves or seek the services of traditional healers.

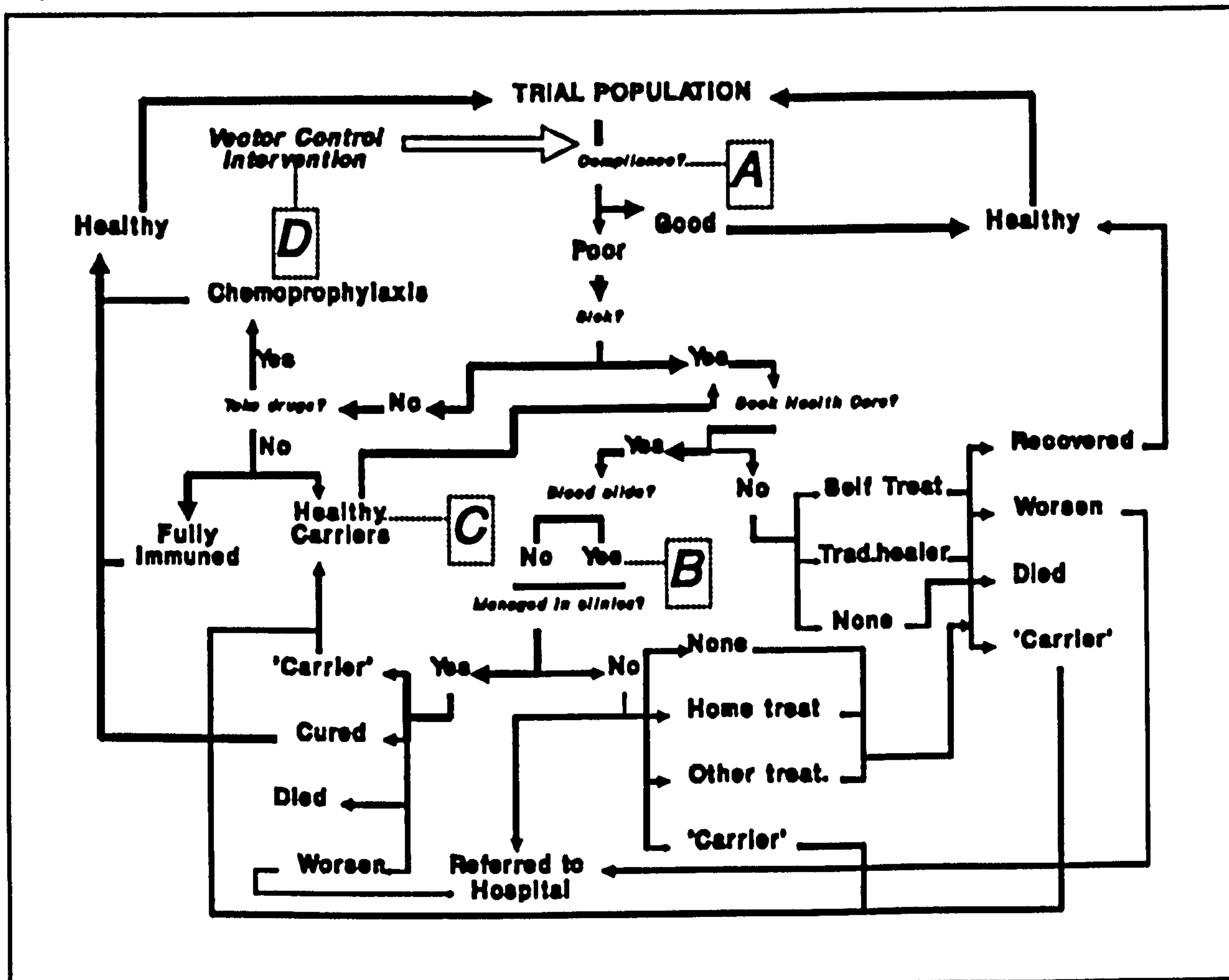
Those that seek care from health workers either have blood slides taken for malaria parasite examinations or not. A well developed standard policy and practice of over two decades is that all fever cases, and those ill, suspected of malaria, are to have blood smears made and microscopically examined for parasites. This has been practically implemented right down to primary health care workers and PCD volunteers. Recent observations indicated as over 92% people attending with fever of complaints suggestive of malaria (Briese, *et al*, 1989) had a blood smear taken. The only time this was not done was when there was no clean glass slide or no disposable lancet available. It was also an established standard practice and policy that only clean slides and disposable lancet be used as an important preventative measure against infections, in particular hepatitis B and human immunodeficiency viruses.

The smear is sent to the nearest laboratory for examination under Giemsa stain and the result later sent back to the original health worker. Records of all positive cases were made, giving specific and clear details of the patient by regions and villages and also other vital information regarding the smears. All these are sent by weekly returns to provincial and national headquarters. This outcome, (B), positive malaria by PCD mechanism, gave information on *incidence of malaria infection*.

Of those that seek health care, some are managed in the clinic either as outpatients, or inpatients when they are too ill. Those that are not treated at a clinic are either treated at home, or are so ill as to have been referred to hospital. However recent studies confirmed (Briese, *et al* 1989) that almost all those seeking health care at a clinic were given some form of treatment at the clinics and aid posts. Those that are treated at clinics have four options. The majority are



**Fig.2: The Algorithm of Selected Outcomes in the study**



### Key

- A - Level of compliance**
- B - Changes in malaria incidence**
- C - Changes in malaria prevalence**
- D - Impact on vectors**
- Cost of interventions and evaluation.**

expected to be radically cured and would thus reconstitute to the healthy population, but some, even though apparently healthy would still have parasites in their blood. This group contributes to a '*carrier*' state till parasite clearance is achieved with the next full course of chemotherapy or development of full protective immunity which will revert them into a non-infected state, subsequently contributing to the healthy population. A few might have become so ill and are therefore referred, while even fewer might die.

**Now to go back to the beginning again. There would also be many who are not ill even**

though they do not fully comply with the interventions. A small group may have taken chemoprophylaxis. Pregnant women, splenectomized individuals, those who have been taking steroids because of artificial heart valves or who have received kidney transplant, and in some cases some infants. Apart from the pregnant women they constitute a minute proportion of people in communities. The main group taking chemoprophylaxis are foreigners and they are mostly in urban areas.

Consequently the majority in this group are either fully protected by a high level of immunity, or are indeed '*healthy carriers*', carrying the malaria parasites without apparent clinical ill health. This group, '*healthy carriers*', (C) also includes those who become so after their malarial illnesses were not adequately treated. This outcome was measured to provide information on the *prevalence of infection*.

Finally in the conceptual framework on the sequence of events in this study, the impact of interventions to changes in the malaria vectors, as well as operational costs of interventions and processes were measured (D).

## 2.7 THE INTERVENTIONS.

### 2.7.1 Justification

The two interventions, DDT residual house spraying and permethrin treated bednets were selected because they aimed at reducing malaria incidence in the community by affecting transmission when applied on a wide scale. They have the same dimensional goal which is of principal public health interest. DDT residual house spraying has been used on a national scale since 1958 and unless a more appropriate and effective alternative is found, may continue to be applied despite technical, administrative and the social problems which have been encountered. Permethrin treated bednets have been field tested (Kere, *et al* 1992) and have appeared to be the

current best potential alternative. If it is confirmed to be the more effective by this study, it is to be adopted on a national scale. Large scale community based studies are expensive and it would not be justified to study such an intervention if it was not to be adopted in public health programmes.

### **2.7.2 DDT Residual House Spraying.**

Residual house spraying with DDT was carried out in communities selected at random. It was applied by supervised teams of trained spraymen, using Hudson X-pert sprayers. All wall surfaces of houses in villages were sprayed at a dosage of 2 gm/m<sup>2</sup>. All sprayer nozzles were regularly checked and calibrated so that the spray at an average distance of 18 inches between the nozzle tip and surface deposited the insecticide equally on the surfaces.

The walls, that were made with thatched materials were, sprayed with 75% wettable, dispersal powder, (75% w.d.p.) DDT, and for surfaces of permanent materials 25% Emulsion Concentrate (EC) was used as standard practice. The insecticide was mixed with water at the village and all structures were sprayed twice a year, making four cycles during the study period. Any remaining mixture left over after a day's work in a spraying cycle was either stored properly, to be used the next day, or was discarded on dry ground away from rivers or streams.

### **2.7.3 Permethrin Treated Bednets**

The bednets used were of Philippine polyethiline monofilament and Thailand nylon multifilament types, obtained through the support of WHO. Three sizes of the Philippine type three were used, 1.65mx0.7mx1.5m as single net, 1.95mx1.0mx1.5m as double net, and 1.95mx1.95mx1.5m as family size net. Two sizes of Thailand types were used, 1.95mx1.0mx1.5m for single/double and 1.95mx1.95mx1.55m as family size. Since the blue/green Philippine type



had been introduced in some communities and been found to be quite durable and very well accepted, they were first distributed and only when these ran out were the Thailand type distributed.

The standard distribution was single adults were issued with single nets, adolescents of same sex within same household shared double nets, and married couples without young children. Adolescents of different sex who have nobody to share their nets with were either given the Philippine single type, or a double Thailand nylon type. Family size net was issued for married couple with at least one infant or child.

Permethrin formulation used in impregnation was 50% EC, marketed by ICI (UK), and which was more appropriate in the field. The decision to use 50% EC was in an attempt to standardise the proportions of water and permethrin concentrate required to mix into the correct solution to impregnate bednets of a particular fabric material. Different materials and whether they were of mono or multifilament, absorbed different amount of solutions. Standardization of the strength of permethrin allowed easy calculations in a large scale operation.

All impregnations were done in the villages when bednets were distributed to all members of households according to the previously conducted census. The solution was mixed on site and the treatment done both by technicians and villagers, wearing plastic gloves. The standard technique (Schreck and Self, 1985) was modified for mass impregnation for standard dose of 0.5g/m<sup>2</sup>. The wet nets were suspended horizontally over tightened nylon (fishing) lines and were allowed to drip back into the containers via a large plastic sheet below. Only when they stopped dripping were they placed flat on a mat, bed, or non-absorbent surface such a plastic sheet, to dry horizontally. After the nets were no longer wet they were hung, either inside the house, or in the shade, until completely dry.

This modified technique (Annex B) was developed and found to be more appropriate in

the field to impregnate large numbers of bednets within the shortest period of time. This saved the amount of insecticide used and did not to cause undue fatigue on those carrying out the procedure. Any remaining solution after a day of impregnation was either kept to be used the next day or discarded on dry ground away from rivers or streams. Impregnating equipment also was washed over dry ground away from streams, rivers or creeks.

The standard solutions required for each type of fabric netting materials used is shown in Table 2. Since the plan was to train selected villagers from the outset, the process of measuring the surface area of bednet, measuring volume of solution, uptake by each net and subsequently calculating the correct amount of permethrin and water needed, were done initially at the

**Table 2: Permethrin and water required to impregnate netting materials for the dosage 0.5g/m<sup>2</sup>**

Type of Netting Materials			Amt.50 %EC per m <sup>2</sup> (ml)	Amt.water per sq.m (mls)	Concentration
Source	Fabric	Filament			
Philp.	Polyethyl ine	Mono	1	14	3.33%
Thai.	Nylon	Multi	1	22	2.17%
China & Other	Cotton	Multi	1	130	0.38%

beginning of the procedure in all villages. As some villagers already owned bednets made of cotton material these were also treated. Pieces of material taken from randomly selected treated nets from villages were sent to Army Malaria Research Unit, Ingleburn, Australia for surface dosage assessment. The sampled treated nets were replaced with treated new ones.

Bednets were treated annually based on early studies (Kere, *et al* 1992) but re-impregnation were done by villagers under the supervision of a field technician. If this continues to be actively

done as well as it has been, and the resulting dosages are correct, a positive step would have been demonstrated towards this intervention being relevant and feasible in primary health care.



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# CHAPTER THREE

## MATERIALS AND METHODS:

(Continued)

### The Study Sites in Solomon Islands

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#### 3.1 THE STUDY SITES

The following sites were "*blocked off*" into pairs, based on factors that have been discussed, for this comparative trial in Solomon Islands [Fig.3]. Censuses in the DDT areas were done and updated during DDT spraying rounds. In the bednets areas the census was initially done prior to distribution of nets, and was updated when bednets were distributed.

##### 3.1.1 Pair One: Isabel Island.

This was in the south-eastern end of Isabel island, Isabel Province, has an area of approximately 1020 square kilometres with a population of 6,418, living mainly along the coasts. There were 48 villages in the DDT area with a population of 3113, and 61 in the impregnated bednet area with population of 3305. The DDT area was served by an Aid Post at Poro, 2 Village Health Workers (VHWs) and 4 PCD Volunteer Agents (PCDVAs) during the study. Most of the outpatient services were provided at Buala Hospital which was 25 kilometres away to the west, at the provincial headquarters. The malaria laboratory serving this area was also at this hospital which can only be reached by sea transport. DDT spraying was done on February 1990, July 1990, February 1991 and July 1991. The villages which were selected for follow up were Poro and Kmaga with a total population of 710.

The impregnated bednets area in the southern end was served by Tatamba

rural health clinic staffed by two registered nurses, which provided malaria laboratory microscopic service and an Aid post at Lepi. There were 2 VHWs and 3 PCDVAs serving the area during the study. Bednets were impregnated with permethrin and distributed on November 1989, and were re-impregnated in November 1990 and November 1991. The selected villages which were followed up were Nagolau and Sepi with a total population of 660.

### **3.1.2 Pair Two: Vella la Vella Island.**

This pair was located in an area covering about 1,220 square kilometres in the northwestern part of Vella La Vella island, Western Province.

The DDT area was the western part with 2,955 people living in 69 coastal villages. It was served by Irigilla rural health clinic which has two registered nurses, Aid Post at Varese, two VHWs, and five PCDVAs. DDT spraying was done in February 1990, July 1990, February 1991 and July 1991. The selected villages of Varese and Vatoro with a total population of 450 were followed up.

The bednets area was the northern part with 52 villages along the coasts and a population of 2312. It was served by Boro Seventh Day Adventist rural health clinic, Karaka Aid Post and also partly by Irrigilla rural health clinic. There were two VHWs and six PCDVAs also serving the area. Bednets were impregnated with permethrin and distributed in November 1989 and were reimpregnated in November 1990 and November 1991. Sibilado and Boro villages, with a total population of 501, were followed up.

The malaria laboratory service available to this pair was at Gizo Hospital, located at the provincial headquarter on Gizo island, about 33 kilometres away. It can only be reached by sea transport.

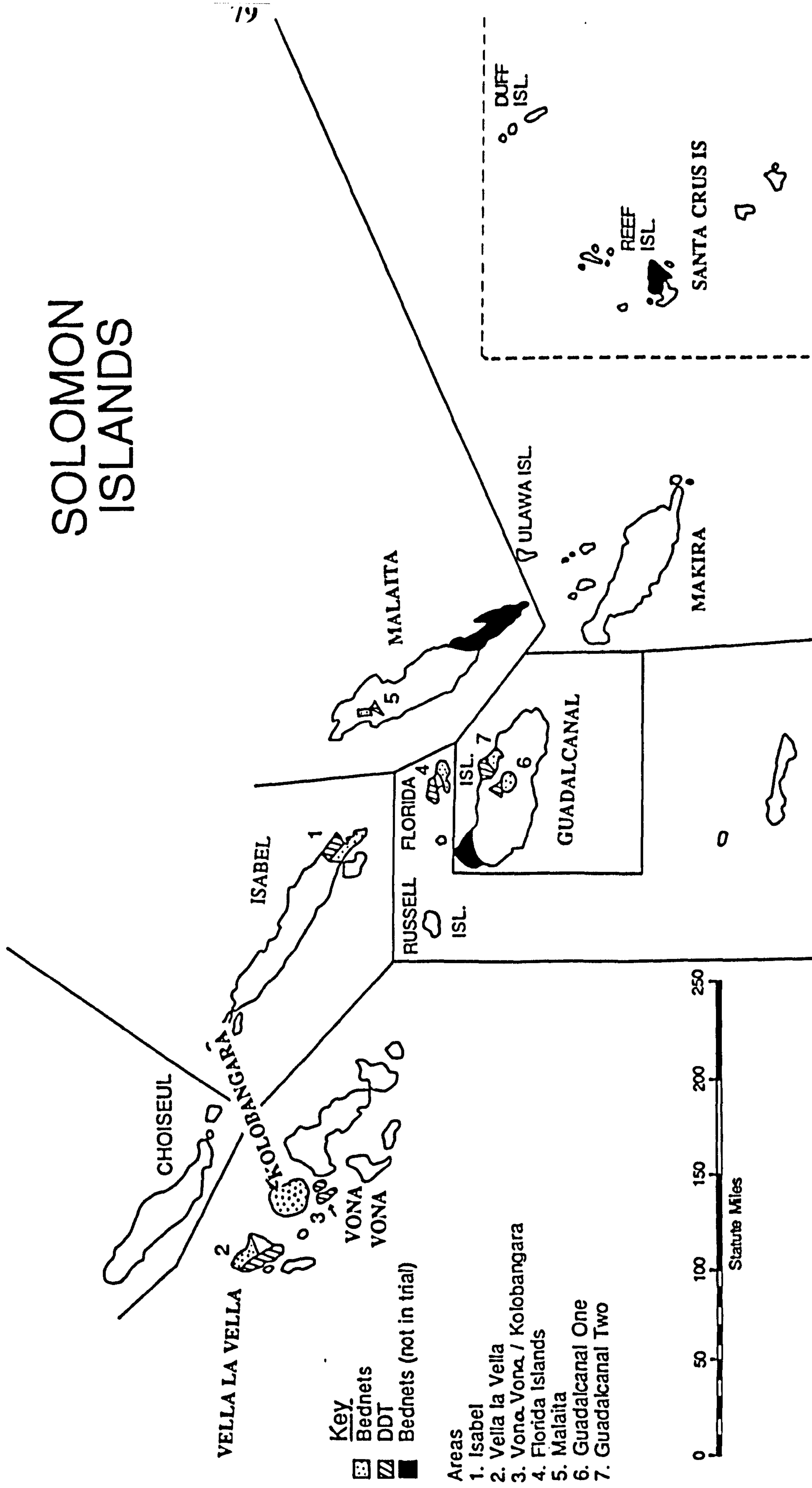


Figure 3. The Multi-centre Comparative Trial Areas, Solomon Islands



### **3.1.3 Pair Three: Vona Vona/ Kolobangara Islands.**

This pair included the islands of Vona Vona lagoon, and the whole island of Kolobangara. Vona Vona which was the DDT area included two major islands and several smaller islands making up approximately an area of 450 square kilometres. There were 32 villages located on coasts of the main islands or in small islands with 2,837 people. The area was served by two Aid Posts; at Boeboe and Buni, two VHWs and five PCDVAs located in villages. The main outpatient service to the area was at the Helena Goldie Hospital, an United Church hospital, located at Munda which was 12 kilometres to the southeast on the main island of New Georgia, and can only be reached by sea transport. The malaria microscopic laboratory serving the area was located in this hospital. DDT spraying was done in February 1990, July 1990, February 1991 and July 1991. Madou and Boeboe villages, with a total population of 550, were followed up.

Kolobangara island, the impregnated bednets area, covered an area of 900 square kilometres. It has 47 villages along the coasts where 2,414 people lived. It was served by Kukundu SDA Church rural health clinic and two private company clinics at Ringi and Poitete. There were three VHWs and four PCDVAs in the area. The main laboratory serving the area was again at Gizo Hospital, 15 kilometres away to the west on Gizo island and could only be reached by sea transport. Bednets were impregnated and distributed in November/December 1989 and were reimpregnated in November 1990 and November 1991. Hunda and Habere villages, with a population of 153, were followed up.

### **3.1.4 Pair Four: Florida Islands**

These islands were 45 kilometres north of Honiara, the capital and consisted of Small Ngella to the east, Big Ngella and Sandfly Islands to the northwest, of Central province. It

covered an area of 840 square kilometres. The only malaria laboratory service available to the whole area was at Tulagi, the headquarter of the province, and which also has the small provincial hospital. It can only be reached by sea transport.

The DDT area included the Sandfly Islands and northern part of Big Ngella, with 62 villages with a population of 4073. The main clinic serving the area was Leitongo rural health clinic, which has two registered nurses, and also Tulagi Hospital. Both can only be reached by the majority of people through sea transport. There was an Aid Post at Tumbila, one VHW and two PCDVAs also served the area. Soso, Tumbila and Ghairavu villages, with a total population of 320 were followed up. DDT spraying was done in February 1989, July 1989, February/March 1990 and July 1990 during the study.

Small Ngella with 48 villages was the impregnated bednet area. It was served by two Aid Posts including one at Siota Secondary School. The main outpatient service was provided at Tulagi Hospital which again can only be reached by sea transport by the majority of people in the area. There was one VHW and three PCDVAs serving the area. Bednets were introduced in 1987 but due to an interruption by a Mass Drug Administration campaign in early 1988, the study proper was started in October 1988. The bednets were reimpregnated in January 1989 and January 1990. Siarana and Nata villages, with a population of 183 were followed up.

### **3.1.5 Pair Five: Malaita Island**

This pair was 3 kilometres inland from the west coast of central Malaita, Malaita province. The area, where A.punctulatus was the main vector covered an area of 450 square kilometres with ten villages. The DDT area included six villages with a population of 634 and impregnated bednets, four villages, with a population of 435. DDT spraying was done on

March 1989, July 1989, March 1990, and July 1990. Laguwata with a population of 120 was followed up in DDT area. Permethrin impregnated bednets were distributed in November 1988 and were re-impregnated in November 1989 and November 1990. Buiano village with a population of 110 was followed up.

The whole area was served by only one VHW and 2 PCDVAs and the main outpatient service was available at Fauambu Anglican Church clinic, located only 3 kilometres away along the coast. Malaria laboratory service was only available at Kilu'ufi Hospital, at Auki, the provincial capital 31 kilometres away to south and can be reached by motorable road.

### 3.1.6 Pair Six: Guadalcanal One

This was another pair where A.punctulatus was the main vector. It was located in central North Guadalcanal, 30 kilometres east of Honiara the capital, and 10 kilometres inland. The area was served by Binu Rural Health Centre which was located 4 kilometres north of the area along the main road, and Solomon Islands Plantation Ltd. a private clinic at Ngalibiu, also located along the main road which runs parallel with the coast but 5 kilometres inland. Both provide malaria microscopy services.

The DDT area covered 20 villages with a population of 959. DDT spraying was done in February/March 1989, July 1989, February 1990, and July 1990. There were four PCDVAs serving the area. Bebe, Boromole and Sole villages, with a population of 198 were followed up. The bednets area covered 23 villages where 860 people lived. These villages were only served by three PCDVAs. Impregnated bednets were distributed in October/November 1988 and were re-impregnated in October 1989 and October 1990. Bemuta, Vasakiki, and Putukoli villages, with a population of 186 were followed up.



### **3.1.7 Pair Seven: Guadalcanal Two**

This pair was located on North Guadalcanal about 35 kilometres east of Honiara and covered an area of approximately 510 square kilometres. DDT area covered 29 villages with a population of 987. It was served by an Aid Post at Ruavatu Secondary School, one VHW and three PCDVAs, and was also served by a rural health clinic at Aola which was 5 kilometres to the east of study area, and Binu Area Health Centre. DDT spraying was done in February 1990, July 1990, March 1991, and July 1991. The village of Tasimboko with 94 people was followed up.

The bednets area was the western part and consisted of 22 villages with 659 people. The main clinic again was Binu Health Centre which also provide malaria microscopy service. There were 4 PCDVAs and one VHW also serving the area. Bednets were impregnated and distributed in March 1990 and retreated in January 1991. Komosou village, with a population of 132, was followed up.

## **3.2 THE EPIDEMIOLOGY AND CONTROL OF MALARIA IN SOLOMON ISLANDS.**

The seven pairs of study communities, which have been discussed, are located in several parts of Solomon Islands (Fig.3). To assist in further understanding these study areas it is necessary to discuss some general information about the Solomon Islands, in particular the existing health care services, epidemiology of malaria, and malaria control activities.

### **3.2.1 Geography and Government.**

The islands stretch 1,400 km from Papua New Guinea in northwest to Vanuatu in southeast between longitudes 154°E to 162°E, and latitudes 5°S and 12°S. The six main islands (Figs.1 and 3) and hundreds of smaller islands make up the total 27,556 sq.km of total land

mass within over 900,000 sq.km of territorial area. The islands have central mountains of past volcanic activity and narrow coastal plains where most inhabitants live. The stretches of sandy beaches are often interrupted by thick mangrove swamp estuaries that are excellent breeding sites for mosquitoes. There are several rivers, creeks, streams, ponds and ox-bow lakes that also provide extensive breeding sites.

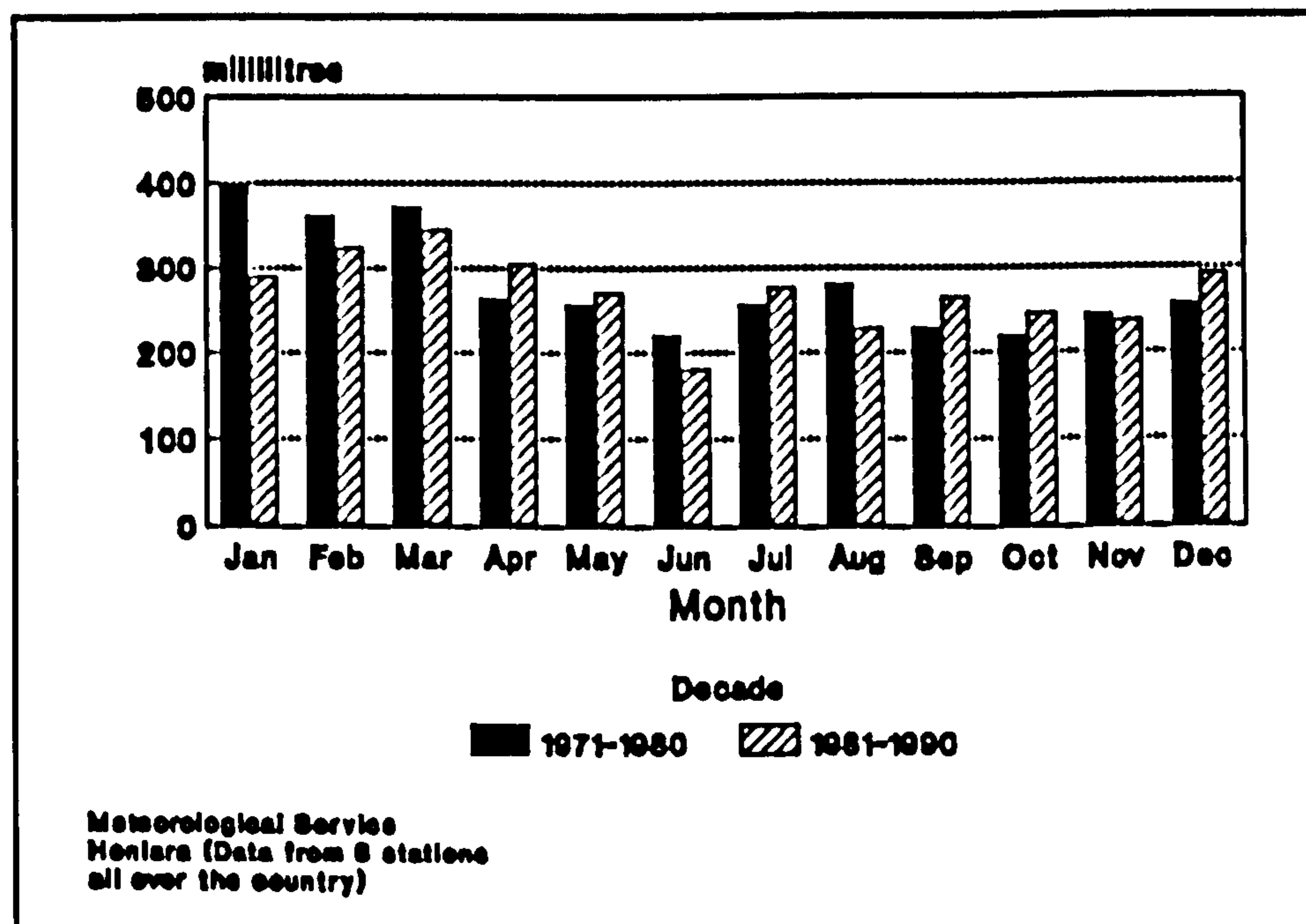
Solomon Islands was probably settled 4000 - 5000 years ago by Austronesians, a neolithic people from South-east Asia. The first European explorer to visit was Alvaro de Mendana, a Spaniard who visited in 1568 and gave the islands its name. Britain declared the islands a "*protectorate*" in 1893 to protect the islanders from '*blackbirders*' who kidnapped the more able men to work on plantations in Queensland and Fiji. The Solomon Islands, which gained independence in 1978, were the site of some of the fiercest battles during World War II.

The country is governed by a National Parliament, elected every four years, and, is located in the capital, Honiara, Guadalcanal. There are nine local governments; eight provincial and one Urban Municipal Authority, whose assemblies are similarly elected. Health care services, devolved in the early eighties, are the responsibilities of the provincial authorities including Honiara Municipal Authority.

The climate is oceanic tropical with little variation in temperatures between 35°C and 21°C. The daily average is 28°C, and temperature may drop to 18°C at mountain tops some of which rise to 2000 to 2400 metres high. The average annual rainfall is 3,500mm, occurring mostly during November to April (Fig.4) when northwesterlies blow. This period, a complete calm of low pressure, may be suddenly interrupted by tropical cyclone bringing heavy rainfall with strong winds which may cause extensive structural damage. The period between April to November, is drier. Average relative humidity is 80%, with ranges between 60% and 90%.

The climate is suitable for stable malaria transmission.

**Fig.4. Average monthly rainfall, 1971-80 and 1981-90, Solomon Islands.**



### 3.2.2 Population and Vital Statistics

Solomon Islanders live in coastal or inland villages on their tribal lands practising shifting subsistence agriculture. The first national census was in 1959 and this was later followed by proper National censuses in 1970, 1976 and 1986.

Crude Death Rate (CDR) declined to the currently estimated 10 per 1000 population. Infant Mortality Rate (IMR) dropped from 151 per 1000 livebirths in 1959 to 38 in 1986. The population growth rate increased from 0.97% in 1959 to 3.5% in 1986, one of the highest in the world [Table 3]. The current estimated population, based on 1986 National Census is 320,000, of which 85% live scattered in rural areas. 95% are ethnically Melanesians and the rest Polynesians, Micronesians, Chinese and others. The population is young with 47% under 15 years, 48% 15 to 59 years and 5% 60 years and older. Crude Birth Rate (CBR)



**Table 3: Demographic changes by national censuses.**

Census	Males	Female	Total	AGR*	IMR**
1931			94,066		
1959	65,532	58,544	124,076	0.97	151
1970	85,179	75,819	160,998	2.58	75
1976	102,808	94,015	196,823	3.4	46
1986	147,972	137,204	285,176	3.5	38

\* - *Average Annual Growth Rate (%)*

\*\* - *Infant Mortality Rate*

Source: National Census Office, Ministry of Finance.

**Table 4: Present demographic data of Solomon Islands (Based on 1986 census)**

Estimated population	320,000
Age-Groups 0 - 14 yr	47%
15 - 59 yr	48%
60 yr plus	5%
Sex ratio [M/F]	108/100
Est. CBA* female population,	79,000
Fertility Rate	6.4
Crude Birth Rate	42/1000
Crude Death Rate	10/1000
Infant Mortality Rate	38/1000 live births [M-40, F-36]
Annual Growth Rate	3.5%
Life Expectancy at Birth	Males - 59.9 yrs Female - 61.4 yrs

\* - *Child Bearing Age, 15 to 49 yrs old women.*

is 42 and life expectancy increased to 61.4 years for females and 59.9 years for male. The average Fertility Rate is 6.4. [Table 4]. This steady high proportion of young people

are continually exposed to infectious diseases such as malaria.

### **3.2.3 Education and Economy**

At present only 35,000 (60% of all eligible) pupils enter primary education (MEHRD, 1991) annually. Then only 18.9% of those completing six years of primary education enter secondary schools, and of whom only 6.8% would complete secondary education. Only 120 are sent to overseas tertiary institution each year, and others have to compete for the 1,200 places at the National Tertiary Institution. The large number of dropouts is due to lack of available places in primary schools, 15 provincial secondary schools [secondary schools which only provide up to Form III], and 8 National Secondary Schools. About 3% of the population above 50 years are illiterate [Habu, 1990].

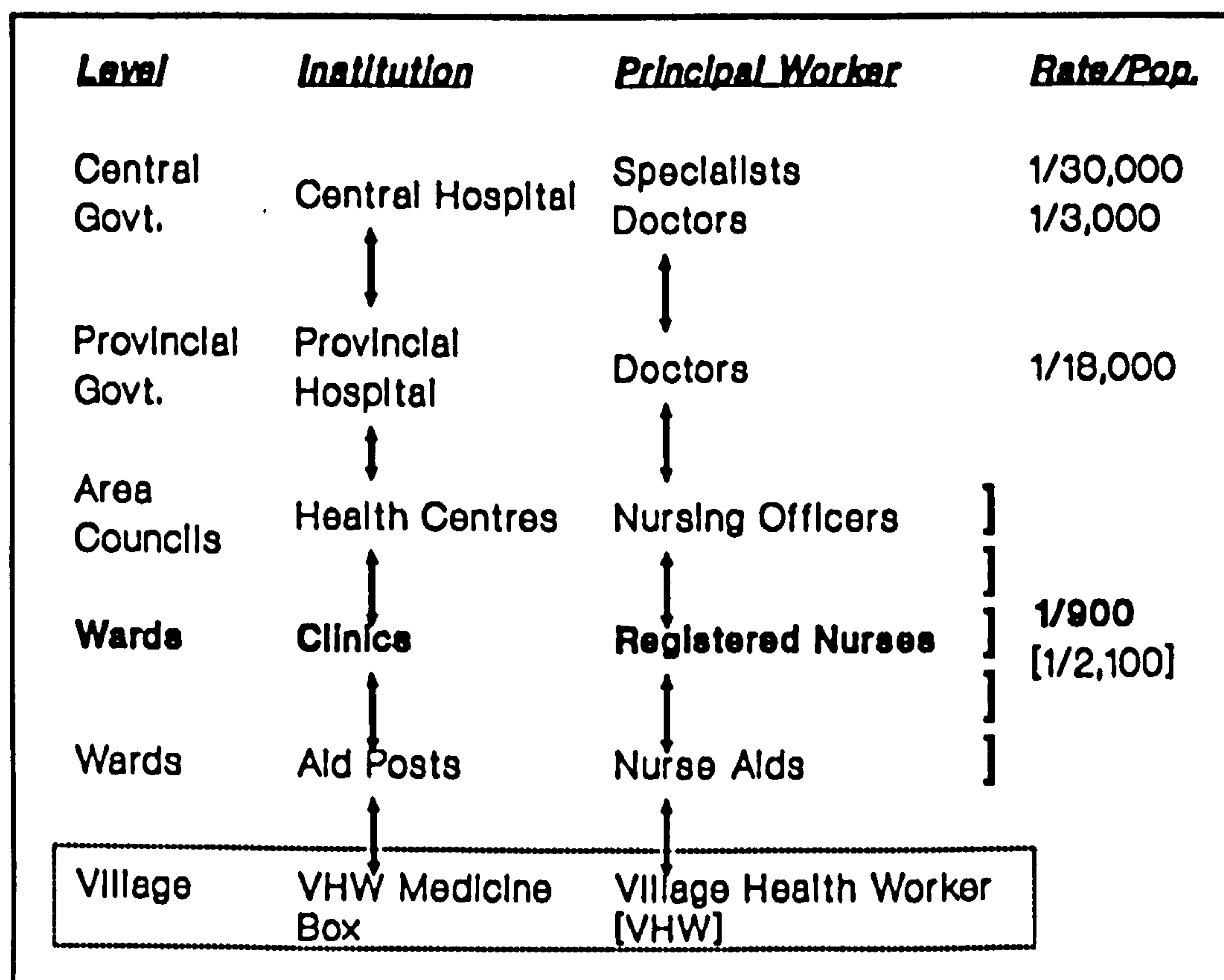
The national economy depends mainly on the export of fish, logs and timber, palm oil extract, copra and cocoa. In 1990 the export earning was US\$60 millions, but imports of machinery, food and others totalled US\$83 millions. There is little manufacturing to offset this imbalance and the average annual Gross Domestic Product (GDP) of US\$226 per capita makes the Solomon Islands one of the least developed countries. The national annual budget of US\$100 millions, with US\$41 millions for recurrent expenses, has to depend on foreign aids for its development component. Population growth is at such a rate that it puts pressure on public services, such as education and health, beyond available resources.

## **3.3 HEALTH SERVICE AND PRIMARY HEALTH CARE DEVELOPMENT.**

Solomon Islands has developed an extensive basic health care service through primary health care. A national health referral system stemming from central to primary health care level [Fig.5] has been established, to serve the widely distributed population. Maldistribution

of both staff and resources exists, with hospitals, especially at central level consuming over 60% of the total national health budget of US\$6.8 millions recurrent, and US\$2.3 millions development. Health budget is only 6.8% of total national budget [MOF,1991].

**Fig.5: National Health Referral System, S.I.**



Health care responsibility of the provincial governments depends on the efficiency of the referral system. The upward referral of problems and patients and central provision of support, advice and resources. The efficiency of the system however depends on imported fuel, oil, and transport and communication equipment which are inadequate with the present acute financial shortages. Health care services are maintained with readjustments and sacrifices especially to promotive and preventive health programmes.



### 3.4 HEALTH CARE UTILIZATION, MORBIDITY AND MORTALITY PATTERNS.

Available data shows high level of health care service delivery and utilization; high health institutional deliveries [Table 5], high level of antenatal services [Table 6] and vaccination coverage [Table 7].

**Table 5. Characteristics of notified births, S.I. 1987-88**

CHARACTERISTICS	1987	1988
Livebirths	9,542	9,565
Stillbirths	124	153
Delivery		
Health institution	8,282	8,404
Village	1,384	1,314
% Village	14.3	13.5

Infectious diseases, mainly acute respiratory infections, malaria, fevers, and childhood diarrhoea [Table 8] are predominant causes of outpatient attendance. Even though only 20%

**Table 6. Characteristics of the MCH services, 1989-90.**

CHARACTERISTICS	1989	1990
Target Population		
Antenatal*	10,913	10,202
Tet.toxoid**	12,868	13,372
Coverage (%)		
Antenatal	94.1	85.6
Tet.toxoid	57.6	61.3

\* - *Estimated number of women pregnant and to deliver that year.*

\*\* - *Estimated number of women pregnant, or immediate post-partum to be completely protected.*

of deaths are estimated to be reported, mainly from hospitals, chronic conditions such as

cardiovascular diseases, malignancies, and chronic respiratory infections are amongst the main causes of mortality [Table 9]. Accidents have become an increasing significant cause of death. At the community level they are components of the primary health care programme, but malaria continues to be the main public health priority. There are several components of services that are being provided, MCH services, EPI, Rural Water Supply and Sanitation, Family Planning and Malaria Control Programmes. All these have adopted various primary health care approaches such as application of appropriate technologies e.g. locally made water pumps, and volunteers as PCD agents.

**Table 7.      Reported immunization (EPI) coverage (%), 1989-90**

<b>IMMUNIZATION</b>	<b>1989</b>	<b>1990</b>
Target population	11,642	11,919
BCG	61.5	78.2
3rd DPT	67.1	62.1
3rd Polio	66.6	61.1
Measles	92.2	53.6

**Source:**      *Health Statistic Unit, MHMS, 1991.*

The health care service is supported by an intensive, extensive and active health education programme. Health education, with the establishment of a Health Education Unit in 1980, has been accorded a top priority. It is a major component of primary health education with the following emphasis;

- a)      how communities fully utilize available services and support all health staff,
- b)      basic relationship between individuals, families and community behaviours to priority and prevailing diseases, and

- c) what possible basic actions can individuals, families and communities undertake to make their community healthy.

**Table 8. Main outpatient morbidities, 1989-90, S.I.**

<u>DISEASES</u>	1989		1990	
	Number	per 1000	Number	per 1000
ARI	156,525	506.9	145,782	457.8
Malaria	66,965	216.9	113,613	354.3
Flu/fever	82,401	266.8	96,354	301.1
Diarrhoea*	12,130	242.6	13,728	248.8
Skin/Trauma	20,614	66.8	30,195	94.4
Conjunctivitis	20,192	65.4	13,919	43.5
Measles	12,770	44.6	354	1.11
VD/GC	621	1.68	754	2.0
TB	496	1.61	502	1.6
Leprosy	15	0.05	11	0.03

\* - In the under fives, rate per 1000 under fives.

Source: Health statistics Unit, MHMS, 1991

**Table 9. Ten commonest causes of reported deaths, 1989-90, S.I.**

CAUSES	1989	1990
Cardiovascular (K)	1	2
ARI		3
Chronic respiratory		4
Diarrhoea		5
Malignancies (X)		6
Malaria		7
Accidents (Y)		8
Perinatal		9
Genitourinary		10
Illdefined	2	1

Source: Health statistics Unit, MHMS, 1991



The principal activities are implemented by all health and health related workers especially at community level, including those of nongovernment organisations such as churches. They are supported by 38 health education workers; four in headquarter and 34 distributed throughout the provinces. The main approach is personal contact by village and community meeting, and school health. Since 1980 several two-day village workshops have been conducted each year to address local problems and utilise local talents as resource personnel.

### **3.5 EPIDEMIOLOGY AND CONTROL OF MALARIA**

#### **3.5.1 Early Antimalaria Activities**

Solomon Islanders have used several forms of traditional remedies to '*cure*' themselves of feverish attacks. The introduction of western medicine in the 19th century included some old antimalarials most of which were highly toxic. The religious denominations assisted the government to extend provision of health care including treatment of malaria. Malaria had been mesoendemic throughout except in certain islands and areas where it was either hyperendemic or hypoendemic. It was said to have killed more troops than bullets during the WWII in Guadalcanal (Black, 1955). Malaria, yaws, tuberculosis and leprosy were rampant in those days [Macgregor, 1957] requiring much government and public attention.

An eradication pilot project was implemented between 1961 to 1964 with DDT spraying. It was remarkably successful [Tewari and Colbourne, 1973]. A Pre-Malaria Eradication Project immediately followed in the next five years with extension of DDT spray coverage. The eradication programme, funded by UNDP, UK, WHO and Solomon Islands, was launched in 1970 and DDT spraying was extended to the rest of the islands. The vertical programme, with technical input from overseas partners, had the following objective,

**Table 10. Pre- and post-DDT spraying mass blood surveys.**

ISLAND	AGE-GROUPS	PARASITE RATES (%)	
		Pre-DDT	Post-DDT
Guadalcanal <sup>1</sup>	All ages	30.0	6.3
	2-9 yr	44.8	0.6
New Georgia <sup>1</sup>	All ages	28.8	2.7
Savo <sup>1</sup>	All ages	39.1	0
Isabel <sup>2</sup>	2-9 yr	42.7	0.9
Makira <sup>2</sup>	2-9 yr	45.4	4.8
Malaita <sup>2</sup>	2-9 yr	44.3	5.0
Choiseul <sup>2</sup>	2-9 yr	37.7	8.8

1 - Macgregor (1966)

2 - Avery (1977)

*"to eradicate malaria from Solomon Islands within a period of time commensurate with resources available"* [Avery, 1974].

The early results were dramatic with reductions in morbidity, mortality, and parasite rates. Regular mass blood surveys in selected large communities (Macgregor, 1966, Avery, 1977) confirmed the dramatic effects [Tables 10].

Notified deaths dropped dramatically in the up to 2 year old per 1000 livebirths [Fig.6]. In areas where DDT spraying was not yet carried out, including Malaita, the average death in this age specific group was 130, till 1968 (Macrae 1979) when sharp drops became comparable to the intervention areas.

By 1971 when spraying covered all the islands the rates became similar. The sharp decline in overall deaths in the same age group [Fig.7] since 1970 can only be attributed to the intensive malaria intervention programme. But malaria cases started to increase in 1976. It quickly spread to islands that were free of malaria, causing epidemics.

Fig 6: Mortality in the up to 2 year old, in different endemic areas pre- and post-DDT spraying.

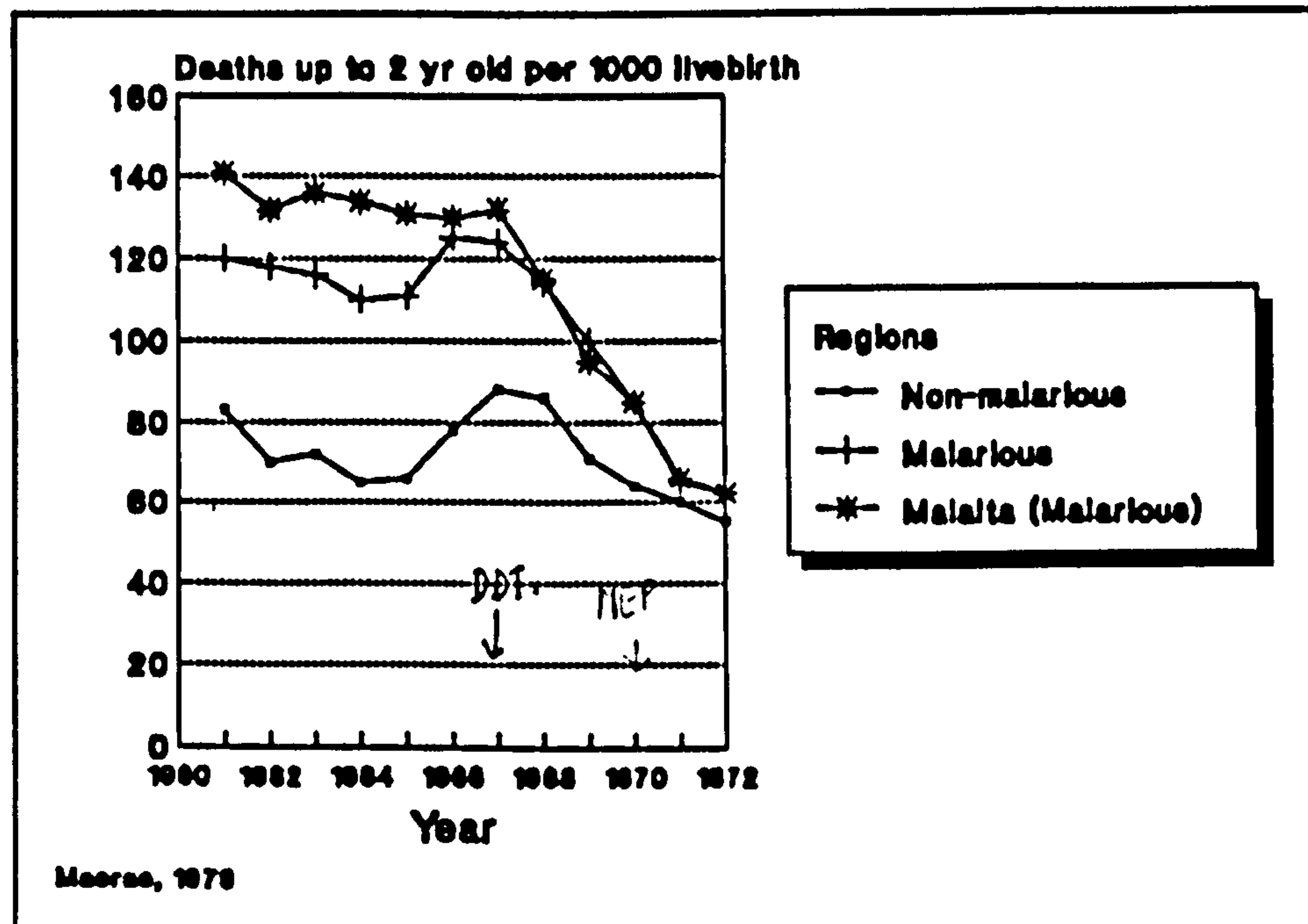
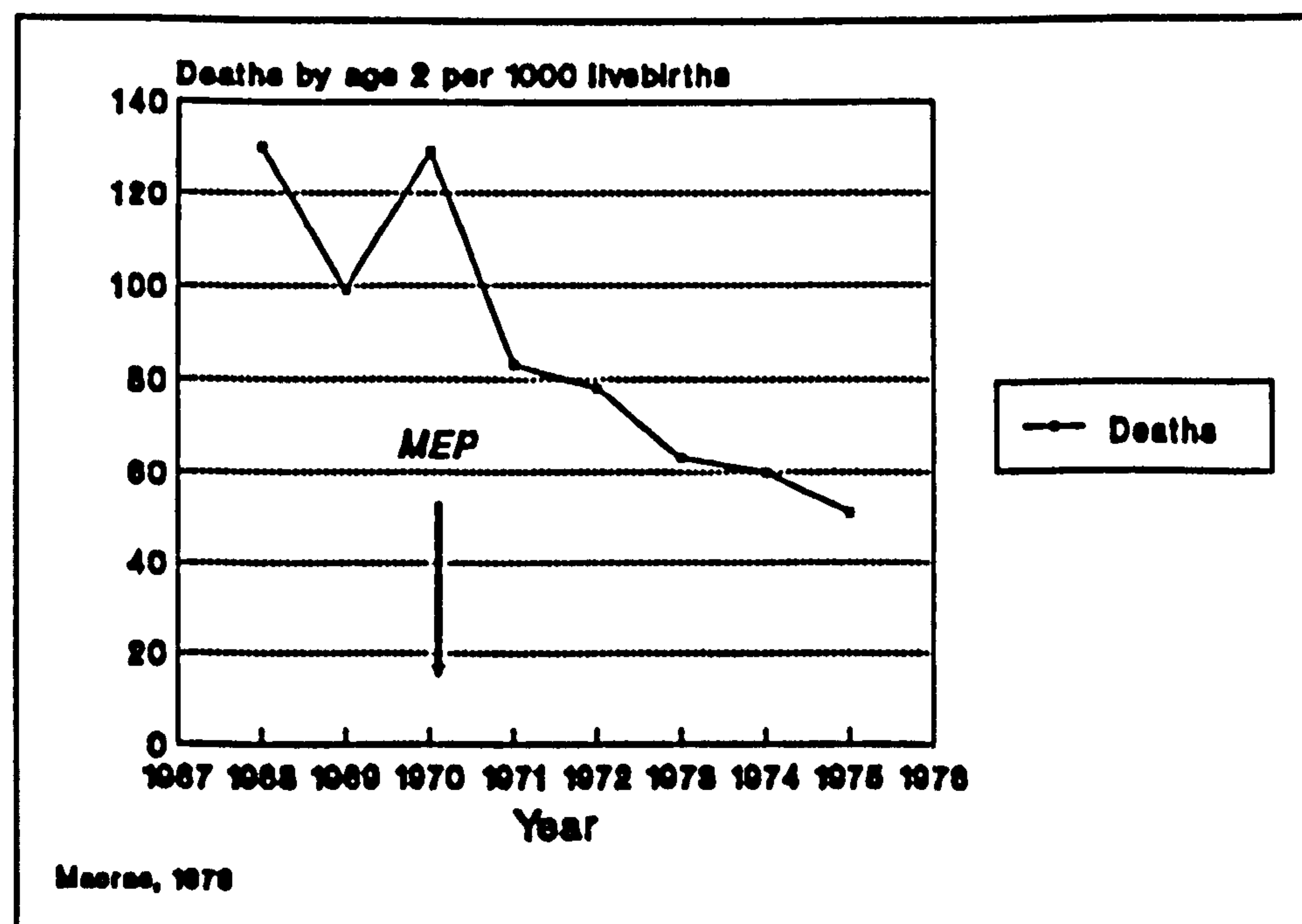


Fig 7: Overall mortality in the up to 2 year old, pre- and post-DDT spraying, Solomon Islands.



In 1980 it became a control programme, with the objective to *"control malaria to a level that it is no longer the main public health problem"*. The specific aims were to reduce malaria



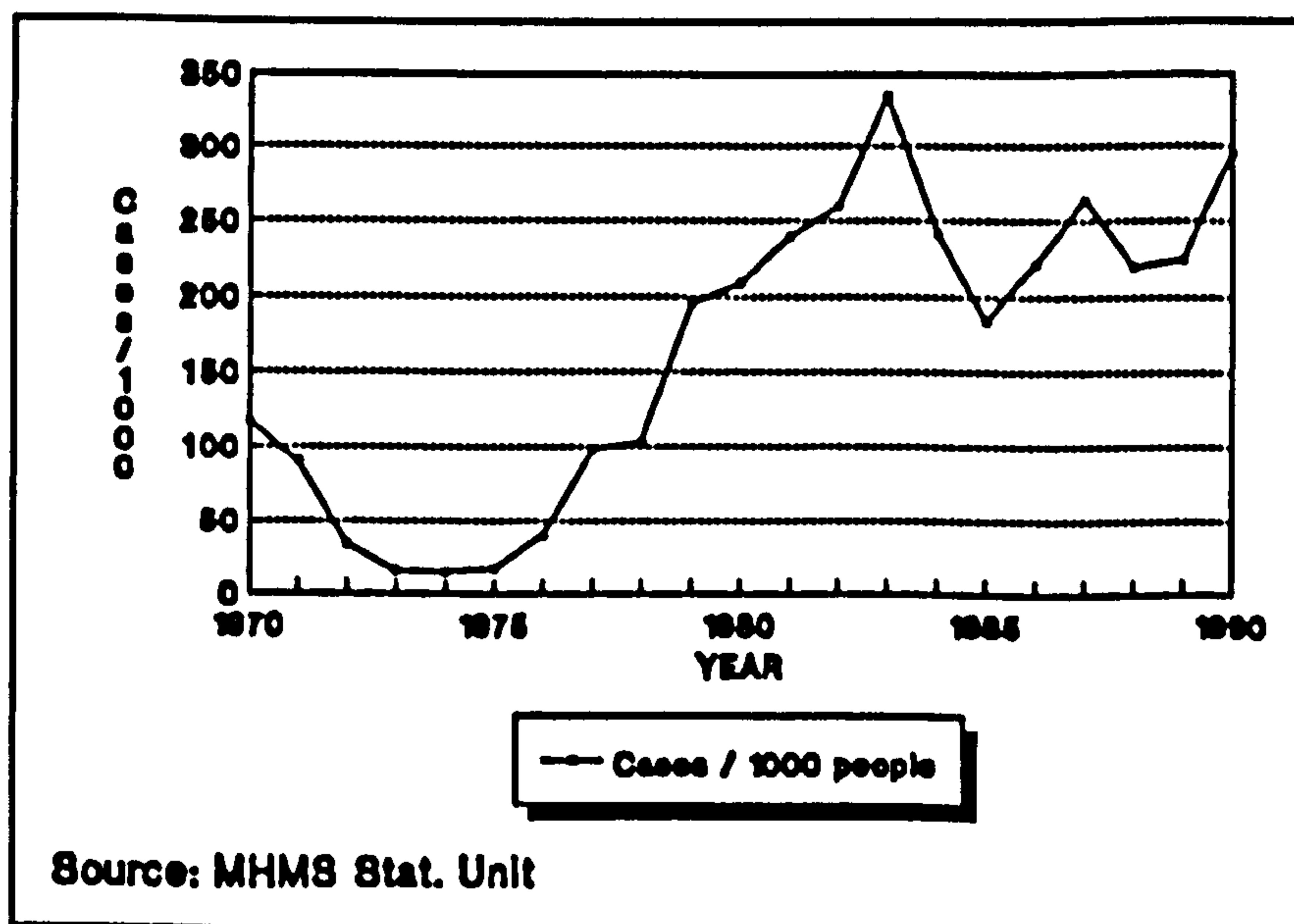
to 80 cases per 1000 population in difficult areas and 40 cases per 1000 in all other areas, even though still maintaining the same strategies.

But by 1989 the malaria situation got worse with rapidly diminishing resources. Therefore the need to determine appropriate strategies for malaria control in primary health care had become necessary.

### 3.5.2 Trend of Malaria Cases.

The malaria cases reported are based on Passive Case Detection (PCD) blood slides microscopically confirmed and this has steadily increased since 1976 to peak at over 300 cases per 1000 population in 1983 [Fig.8]. There was some drop in 1984 and 1985 due to an intensive Mass Drug Administration (MDA) for twelve weeks during peak transmission in central malarious area, that has 35% of national population, but up to 1983 contributed 52% of the malaria cases. The same intense activity failed in 1986 when interrupted by the severe

**Fig.8: Trend of malaria cases, S.I. 1970-1990**

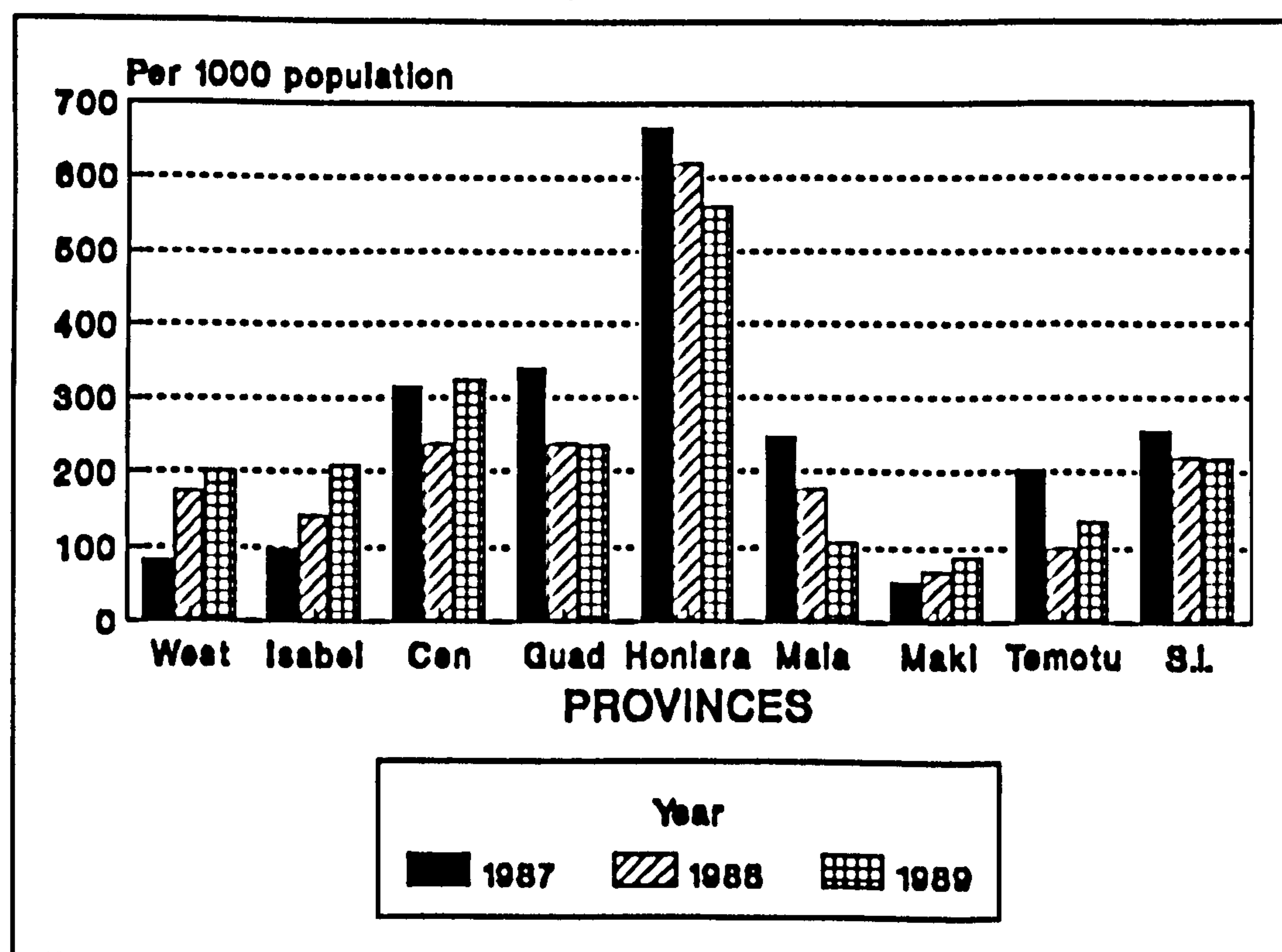


*Cyclone Namu* that caused a national disaster. Further attempts were abandoned as compliance continued to be low. In 1988 cases came down again with reductions in areas where permethrin

treated bednets were used. But with the interruption of other vector control measures after 1988 malaria cases increased in other areas offsetting any declines caused by permethrin treated bednets. The proportion of *P.falciparum* continues to be 60% to 70% of total cases [Fig.11] and which is also influenced by the establishment of chloroquine resistant malaria in 1980.

There are variations in provincial distributions with increases in areas that previously had low number of malaria cases, whilst in highly malarious areas, cases actually declined [Fig.9], including cases in infants [Fig.10]. In Malaita, Guadalcanal and Central provinces where refusals had been highest, DDT failed to have any impact. Therefore since 1986 permethrin treated bednets have been distributed in large numbers in North Guadalcanal, South Malaita and Ngella. The slight declines in 1987 to 1989 [Fig.8] would be due to positive effects of treated

**Fig.9: Incidence of Malaria Cases by Provinces, Solomon Islands, 1987-1989**

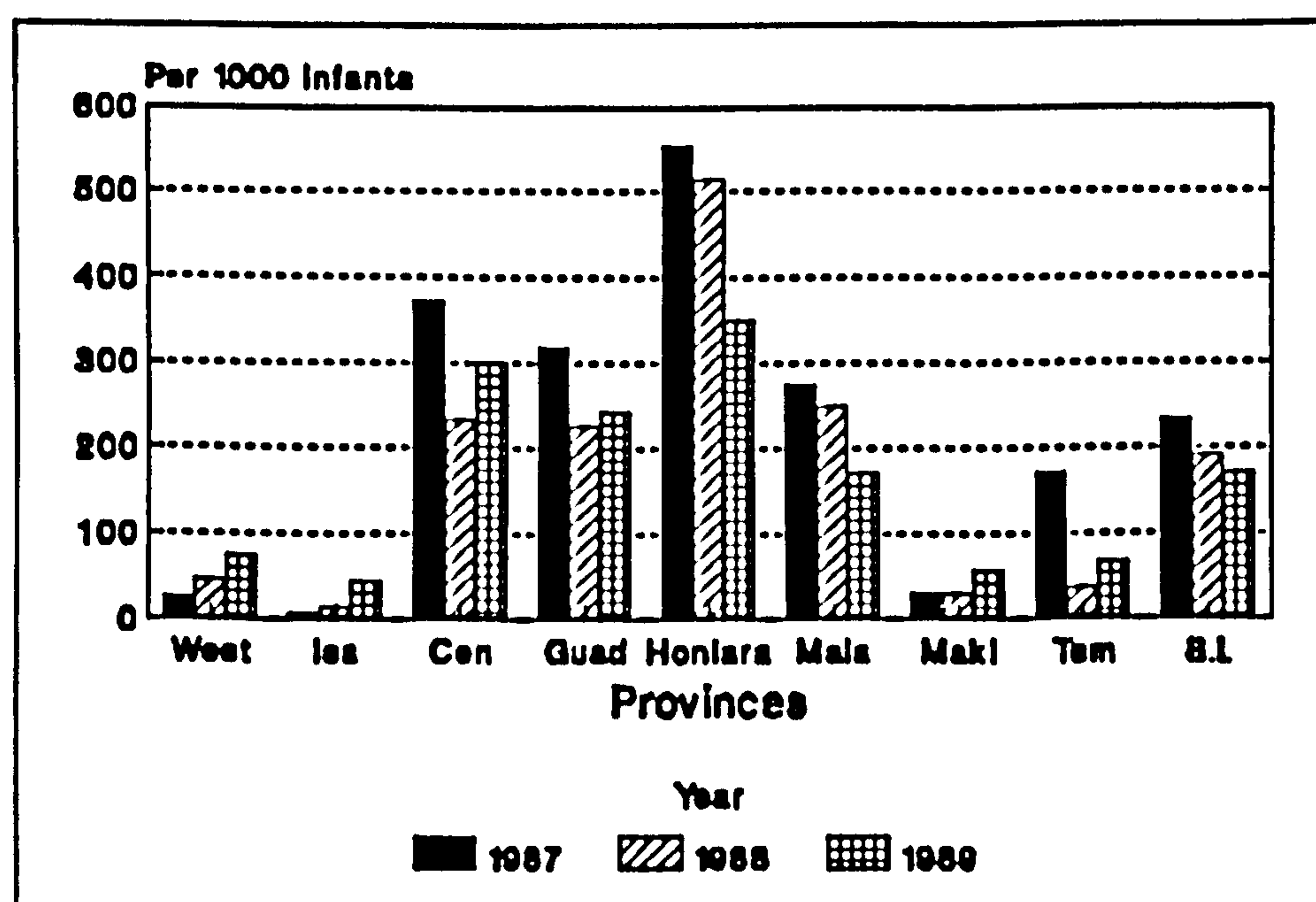


bednets. Since 1988 the Malaria Control Programme encountered serious problems with acute lack of resources and funds mainly with DDT spraying. Inevitably spraying operations practically ceased in all areas (except trial areas). Therefore, cases started to increase in Western, Isabel,

and Makira provinces where compliance had been high. The other possible reason is that 'behavioral' resistance of the vectors to DDT has now been so well established that increases would have happened even if there were DDT spraying. At the end of 1989 further permethrin treated bednets were introduced in these areas and the results form the basis of this thesis.

The distribution varies during the year with the peak transmission generally occurring in the first quarter of the year [Fig.11]. This is the period of the wet season, and also immediately after the holiday season when population mobility is at its highest. During Christmas holiday large number of people stay outside throughout the night carol singing exposing themselves

Fig.10: *Incidence of malaria in infants, by Provinces in Solomon Islands 1987-1989.*



to mosquito bites. Population movement is the strongest determinant of this peak transmission pattern.

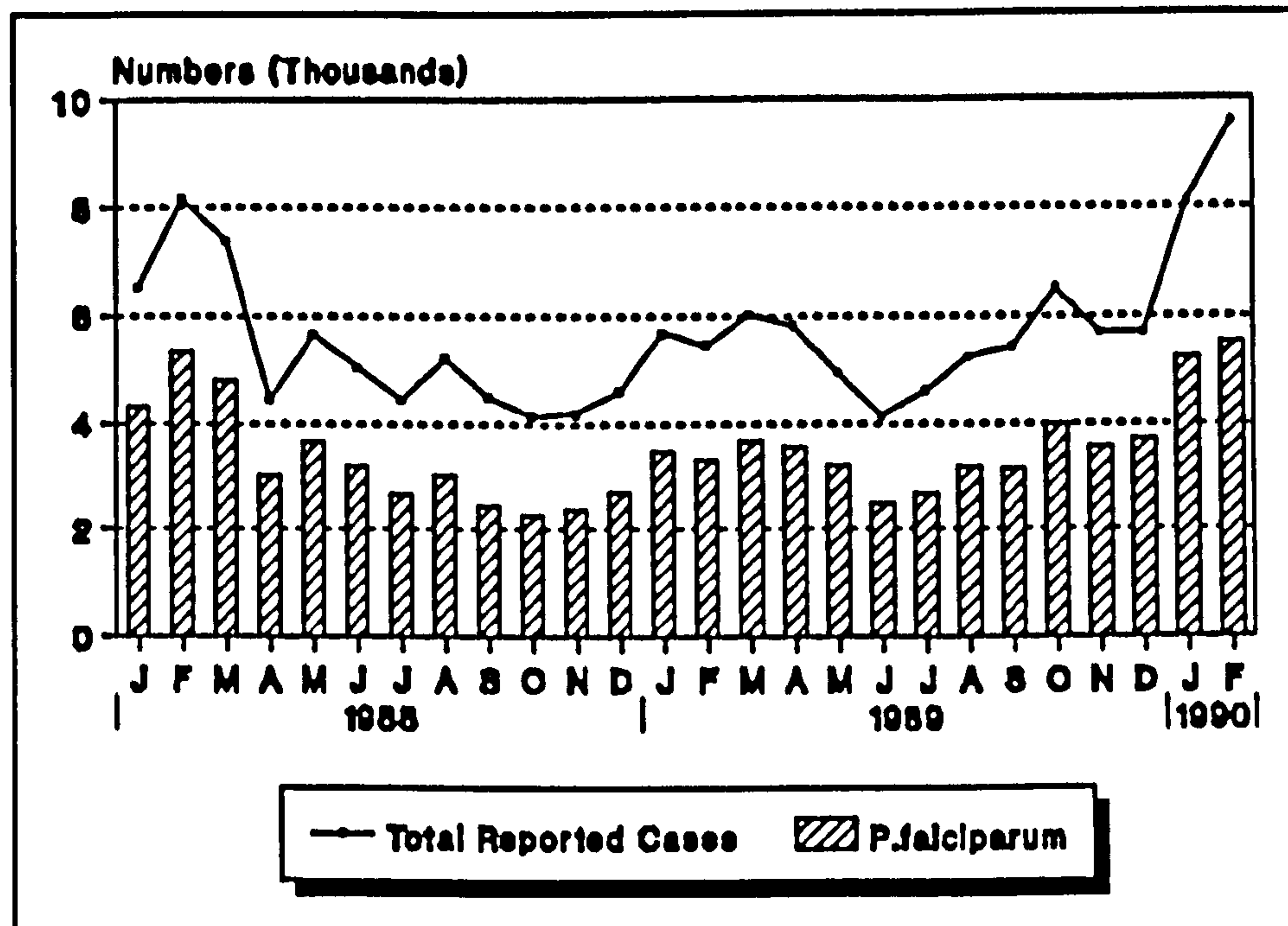
### 3.5.3 Main Vector Control Methods.

#### a. *DDT Residual Spraying.*

DDT spraying till 1988 and 1989 was the main vector control method since 1961. There was a temporary cessation in the late seventies in some areas that were declared "*consolidation*"



Fig.11: *Monthly distribution of malaria cases, S.I. 1988 - 1990(Feb)*



areas according to 'phases' of eradication. Indoor walls of houses were sprayed twice a year at a dosage of 2g/m<sup>2</sup>. Compliance varied from place to place. It was high in Western, Isabel and Makira, low in Malaita and Guadalcanal where most refusals occurred (Paik, 1979) and where most cases occurred [Fig.9].

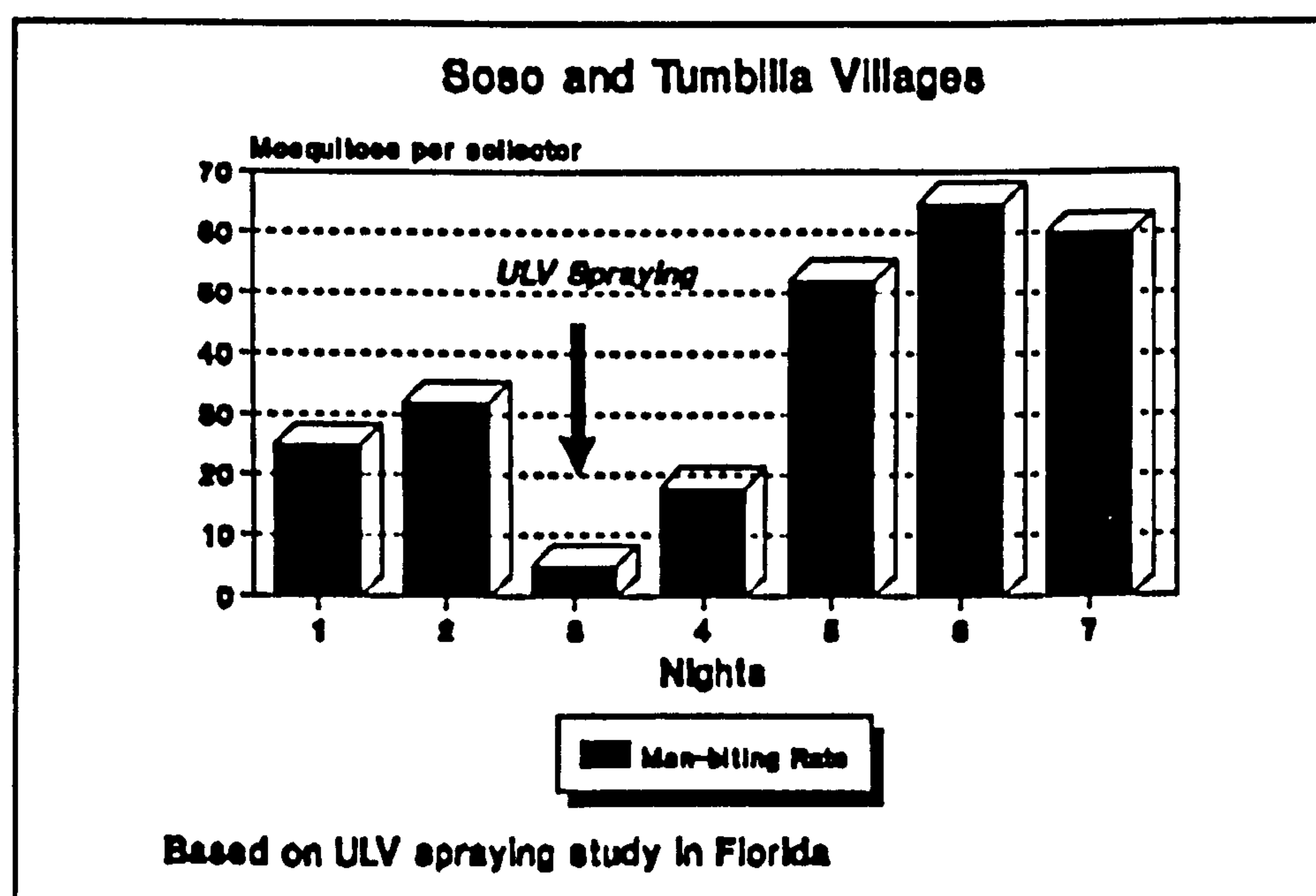
Since 1989 DDT spraying operations became severely disrupted and practically ceased in several places due to lack of funds and resources. This method is incompatible with a programme which is being integrated into general health services. Therefore its use in an integrated system needs to be justified.

**b. ULV Spraying.**

Ultra low volume (ULV) spraying using machines was done sporadically, mainly in urban areas using the organophosphate compound, *malathion*. The impact has not been properly evaluated. In some urban areas larviciding is also carried out. The operation encountered difficulties with poor management, lack of funds and resources. The weekly operation is

expensive. A trial operation carried out by trained villagers in Florida islands, using ULV fenithothion, *Persguard*, resulted in failures. It met with serious problems with supplies, repair, maintenance, management and supervision. Entomologically *A.farauti* increased immediately a few days later [Fig.12] and reinvasion from outside sprayed areas occurred even within two hours. Thus its use in rural communities where villages are surrounded by thick vegetation is very limited, even though it is a useful method in epidemics before a permanent measure is instituted.

**Fig.12:** *Entomological impact of community ULV spraying, Florida Islands.*



### c. *Larviciding*

Larviciding with the organophosphate temephos (*Abate*) has been sporadically done both in urban and rural communities. It again encountered problems with lack of funds and resources, poor management and supervision. A trial using *Bacillus thuringensis israelensis* (*B.t.i*) was conducted in communities in Ngella islands using village volunteers as applicators with some initial success [Ebsworth and Denty, 1989]. But this quickly met similar problems. The

compound, B.t.i. is expensive and so is the weekly operations. Thus larviciding with either compound is left to urban authorities or private companies who bear the cost.

An appropriate method to attack adult female mosquitoes is desirable where the cumulative effect of even a moderate kill on each occasion may prevent the mosquito from living long enough to develop mature sporozoites. But any breeding site which is missed in larviciding would yield adult mosquitoes with a good chance of surviving to a dangerous age (Curtis, 1990).

#### d. Research

Active research is being undertaken for vector control methods, appropriate in primary health care, either alone or in combination with other measures. A juvenile hormone mimic (JHM), *pyreproxifen*, [S11183] which was developed by Sumitomo Chemical, Japan, larvivorous fish *gambusia affinis*, tilapia [*Oreochromis species*] and a guppy species [*Poecilia spp*] are being studied, especially the latter, with community involvement.

The JHM, pyreproxifen, was field tested successfully in both emulsion concentrate (EC) and granular formulations against immature forms of the vectors. A single application is found to inhibit growth of A.farauti with emulsion concentrate (Suzuki, *et al* 1989), and A.punctulatus with granules (Kere, *et al* 1989), for at least three months. Plans are underway for a larger community field trial.

A new insecticide, etofenprox (*Trebon*), developed by Mitsui Chemical, Japan, is currently being field tested for its effect on A.farauti in residual indoor spraying and impregnating bednets. It has a high margin of safety in mammals, with LD<sub>50</sub> for rats of >42,000 mg/kg and is environmentally safe. It is decomposed completely into carbon, hydrogen and oxygen which naturally occur in nature.

The current major research is on pyrethroid impregnated bednets. Early field experiments

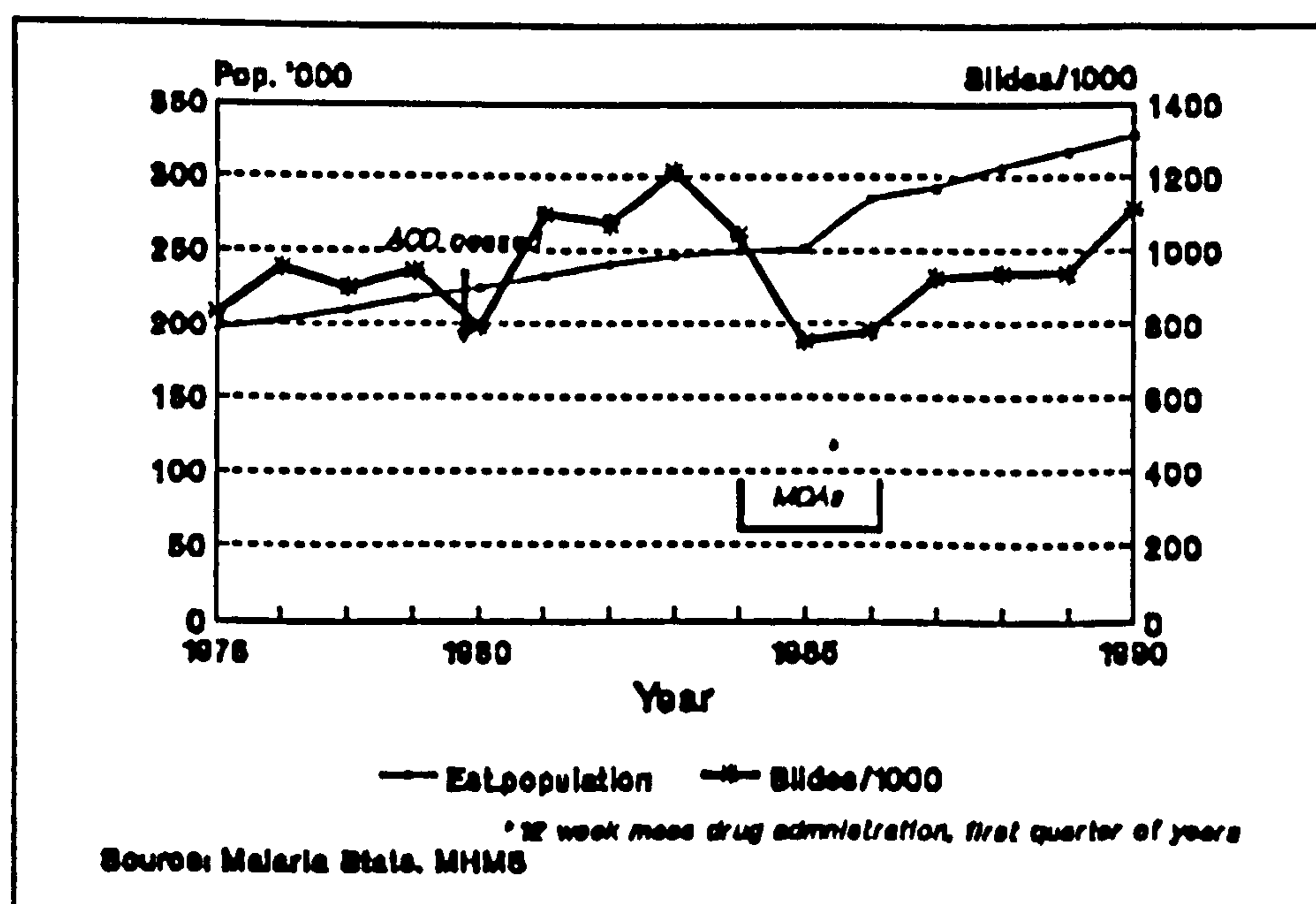


showed potential successes [Kere, *et al* 1992]. It has been tried in a large community based study which forms the basis of this thesis.

### 3.5.4 Case Detection and Management.

Positive blood slides by Active Case Detection (ACD), Passive Case Detection (PCD), and Case Follow Ups (CFU) of treated cases, were traditional diagnostic mechanisms. These were continued till 1980 when only PCD remained as policy for case detection. The blood slides, each with both thick smear, and thin smear fixed with alcohol, are sent to laboratories and examined microscopically under Giemsa stain. However since primary health

Fig.13: *Blood slide rate per population examined for malaria, S.I. 1976-1990*



care has been developed, with extensive PCD agents in villages, the number of blood slides increased even though other forms were discontinued [Fig.13].

The appropriateness of the PCD mechanism in primary health care is being considered on the following points; a) since there is acute lack of resources, the benefit is weighed against the cost of a procedure which needs considerable resources, b) large time interval between

"*blood slide taken*" and "*blood slide examined*" and results may never be received to benefit patients, and, c) health workers appeared to have coped very well with their clinical acumen for patient management. Therefore a simpler and cheaper method to provide the same epidemiological information in evaluating malaria control programmes is desired. This is being addressed in this thesis.

Several regimes of malaria treatment and management are examined and those that fulfil the following criteria have been developed. These are, a) to effectively manage *P.falciparum* the most serious and predominant species and to treat others '*clinically*', b) appropriate and feasible in field condition by an up to a minimally trained primary health care worker, c) cater for drug resistance but safeguard the proper use of second line drugs, and d) the dosage calculated to age-groups which field workers find easier to determine.

The established standard clinical management of malaria is as follows. On clinical suspicion of malaria a blood slide is taken and sent for examination. The patient is treated with three day chemotherapy with chloroquine and one day primaquine as in Schedule 1 (Annex C). If the patient turns up again within four weeks another blood slide is taken, and if it was certain that the patient had taken treatment properly and is not severely ill, treatment administered is as in Schedule II, which includes sulphadoxine/pyremethamine combination (*Fansidar*). Otherwise Schedule I is repeated. If the patient turns up within a week, or is severely ill, he or she is treated with quinine as in Schedule III but supervised mostly in clinics where parental quinine may be considered. It is a policy that where *Fansidar* is included it is to be given on the third day. In cases where there is suspicion of cerebral effect, severe illness with other possible causes, patient not responding to Schedule III, and on evidence of other complications, immediate referral to health centre or hospital is indicated.

All *P.vivax* cases are treated with Schedule I and thus relapses are common. The 14-day

radical treatment was discontinued as a policy and whether to radically treat or not remains with health workers. The conceptual framework for malaria management has been developed [Annex B] and standard management schedules will be modified when new drugs come into use.

### 3.5.5 Parasites and Drug Resistance.

P.falciparum, P.vivax, and P.malariae are the known human malaria parasites in Solomon Islands. In the seventies when malaria was low P.vivax predominated with average ratio of 3:1 (Avery, 1974) or even 5:1 (Avery, 1977) over P.falciparum. P.malariae became very rare. At present P.falciparum is the predominant species making up between 60% to 70% of the cases. The sporogonic cycles of P.falciparum and P.vivax within vectors in ideal conditions are 12 days and 7 days respectively (Macdonald, 1957). Thus an effective vector control intervention that reduces longevity would first reduce P.falciparum. However, as vector control methods have become inefficient, P.falciparum has become predominant.

Chloroquine resistant P.falciparum is established since 1980. It is estimated that almost half of the P.falciparum infections are resistant to chloroquine, mainly of Grade I resistance. Fansidar is used effectively as the alternative drug where resistance is suspected. The increased tolerance of P.falciparum to amodiaquine, which is often substituted for chloroquine in children, is being investigated and there is no suspicion of resistance to other drugs. Halofantrin was successfully studied clinically but its use is limited due to high cost.

### 3.5.6 Vectors and Insecticide Resistance.

The Anopheline mosquitoes transmitting malaria in Solomon Islands are species of the Anopheline punctulatus complex; A.farauti Laveran, A.punctulatus Donitz, and A.koliensis. Both A.punctulatus and A.koliensis disappeared with vector control activities. But whilst A.koliensis

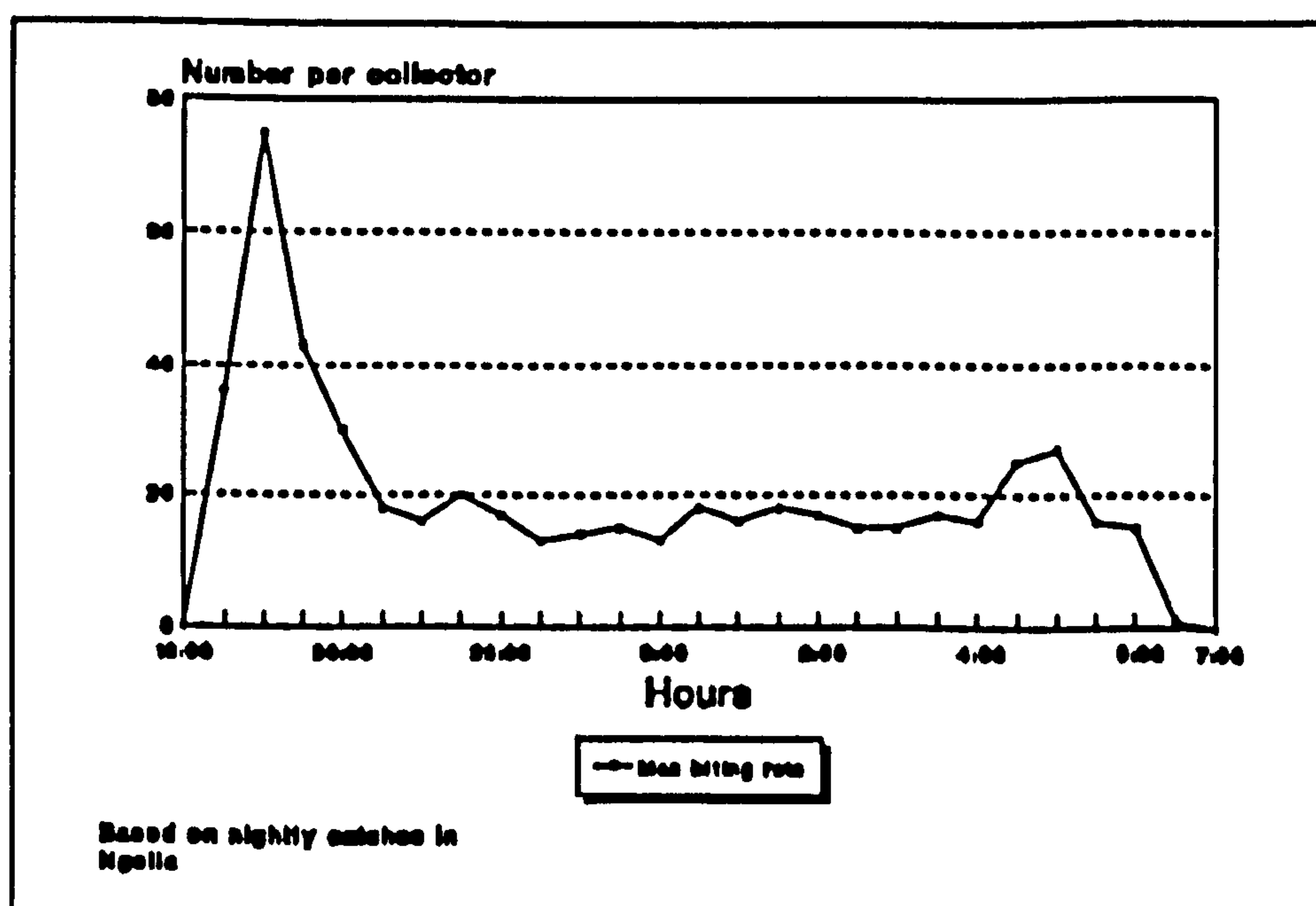


is still very rare, A.punctulatus has reappeared in inland areas. All species, especially A.punctulatus and A.koliensis, with human blood index (HBI) of at least 0.85 (Garret-Jones, 1974), are highly anthropophilic. But it appears that A.farauti would feed on the nearest blood meal if humans are not available.

The sporogonic activity within these species especially for P.falciparum has been very efficient (Burkot 1987). All breed in ground surface water, with A.punctulatus mainly inland in ditches at roadside and wheel ruts on old roads. But A.farauti has also been breeding in highly saline water such as estuaries and swampy areas.

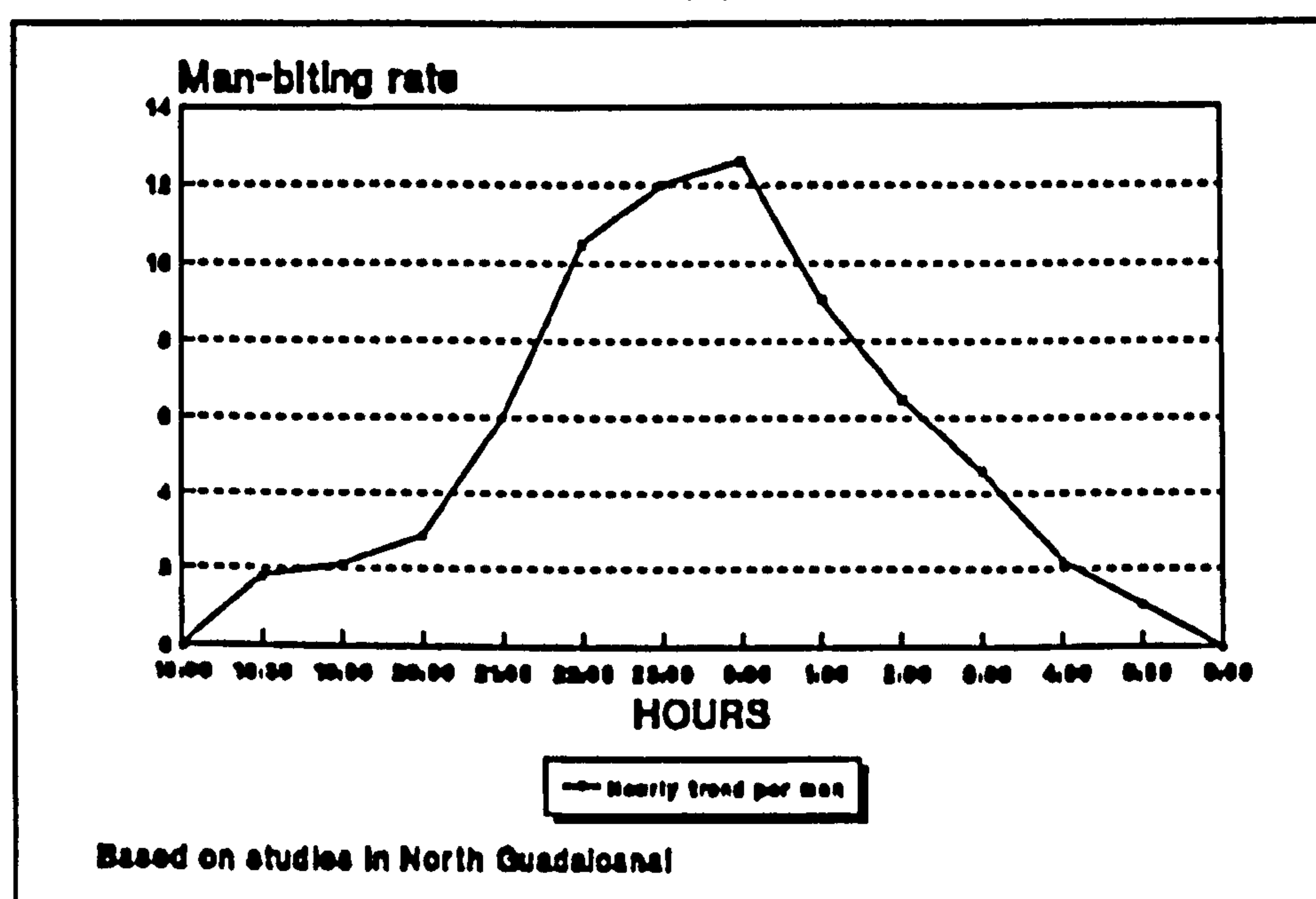
The pressure of DDT changed the behaviour of A.farauti from biting peak at 2200 hrs to 1930 hrs, even after two cycles of DDT spraying (Taylor, 1977). This behaviour has continued in this species (Fig.14) including prominent outdoor biting habit. Even though a lot of A.punctulatus bite outdoor, there is significant indoor biting, and unlike A.farauti, it has not changed its overnight biting behaviour [Fig.15].

Fig.14: *Biting behaviour of A.farauti*



that do enter the house escape outside to rest after feeding, exhibiting exophilic behaviour. This established 'behavioral' resistance maintains high transmission despite continued DDT spraying. It probably also explains why *A. farauti* has not developed chemical resistance, as it avoids contact with the insecticide on sprayed surfaces. Insecticide treated bednets offers better protection as persons are under bednets.

**Fig.15:** *Biting behaviour of A.punctulatus.*



### 3.6 CURRENT EFFORTS AGAINST MALARIA IN PRIMARY HEALTH CARE.

The main efforts in primary health care level is to diagnose and treat malaria before complications develop. Thus PCD and patient treatment are the major responsibilities at primary health care level. Feasible source reduction by village cleaning, proper drainage, cleaning of drains, streams, and proper construction of wells are emphasized at community level. The latter is a primary health care component of the *Rural Water Supply and Sanitation Project*. All these have been the main subjects of the series of regular 2-day village PHC workshops conducted since 1980.

A special study is being initiated to involve communities in raising their own fund for purchasing netting materials and also for sewing bednets. Even though it is still early, the interest

motivated is high. Another study is looking at the feasibility of communities to mass culture, distribute, and use larvivorous fish. The primary health workers, involved in these efforts are mainly under the guidance of the rural clinic nurses who form the main health workers at the primary care level in the national health service [Fig. 5]. They are supported by Nurse Aids trained for a year and posted to areas not easily reached by registered nurses or as assistants to registered nurses. The Village Health Worker (VHW), trained for three months in basic curative care and basic health promotional, preventive knowledge and skills, is placed in villages not easily reached by nurses. The VHWs come from the same community and under the authority of the community committee. The turnover of these primary health care workers, trained since 1977 is high since they are volunteers, and do not often receive the support required. Lately, however, many continue working, as in 1990 157 [MHMS, 1990] are estimated to be still active. Some microscopists are trained and are placed under the responsibility of certain well organised village communities, firms located in rural areas and church organizations. This is still limited due to the unavailability of adequate equipment and resources. But those that have been trained have supported primary health care malaria diagnostic and management activities.

A new category of primary health worker introduced since 1980 by malaria control programme is PCD Volunteer Agents (PCDVA). These are retired health workers, government workers, teachers in village schools and any willing, trusted and intelligent villagers. They spread over villages in rural areas performing mainly case diagnosis, taking blood slides and administering treatment. All are trained locally and set up in their villages by provincial malaria officers. They, with the registered nurses, nurse aids and VHW, form quite an extensive network of PCD agents. The exact number of these primary health care agents is uncertain as being volunteers they "*come and go*" whenever they wish. But judging from the amount of blood slides received for examination, many of them continue working.



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## CHAPTER FOUR.

### RESULTS, ANALYSIS AND INTERPRETATIONS.

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#### 4.1 DATA ENTRY AND MANAGEMENT.

All computer entries, storage and management of parasitological data, were done with DBase III plus (Ashton-Tate, 1986). The analyses were done with SSPS (version 4.0, SSPS Inc.1990) and Epiinfo (version 5.0, CDC/WHO, 1991) statistical packages. The management and analysis of social questionnaire survey data were done initially with SurveyMet (version 1.61, Henry Elkins, 1985-1988) statistical package, but were later exported to, and analyzed by Epiinfo. All graphics were performed with Harvard Graphics (version 2.10, Software Publishing Corp. 1987) and the word processing with WordPerfect (version 5.1, WordPerfect Corporation, Utah, 1989).

#### 4.2. THE PREVALENCE OF MALARIA INFECTION.

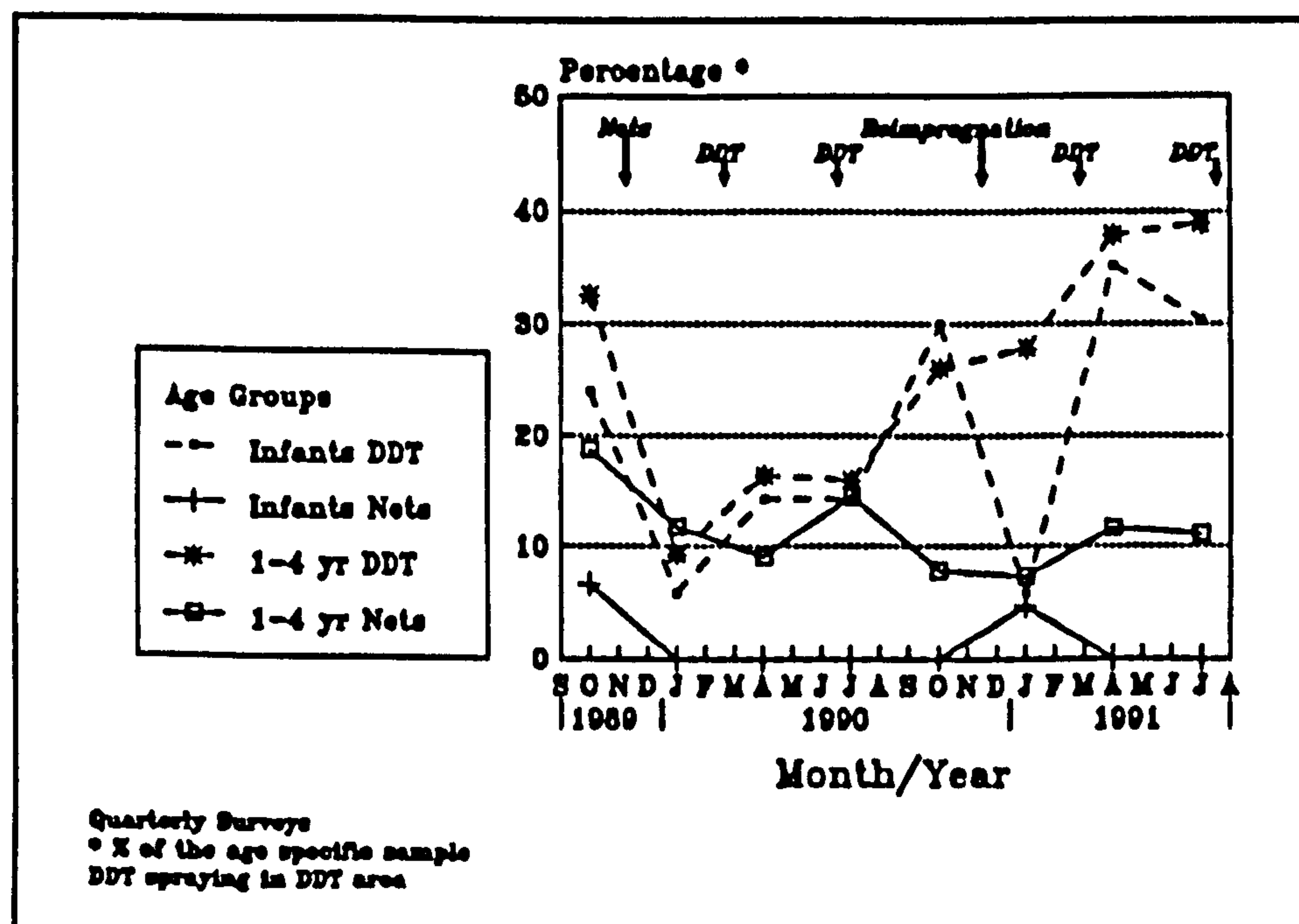
The tables of prevalence survey results are in Annex E.

##### 4.2.1 Isabel Island

In Isabel island the prevalence in under fives, infants and 1-4 year old in both groups are presented in Fig.16. In both groups the rates in the DDT areas started higher than in the bednet group, but dramatically fell in the second survey. There were steady increases thereafter except infants whose rate dropped to 5.9% in the sixth survey but increased again at the end of the study. The prevalence in the bednets group similarly fell with that of infants maintained at zero prevalence except in the sixth survey when one case of P.vivax was diagnosed. the low prevalence in the 1-4 year old was maintained. The low prevalence in the second survey, in all

age groups, was due to a misunderstanding in the field. Primary health care workers in the field undertook a mass drug administration in the survey villages immediately prior to the second survey, as a remedial measure taken on the results of the first survey. Immediate actions necessary were taken to prevent such error throughout the study after this episode. However it also allows some idea of existing transmission potentials in the intervention areas.

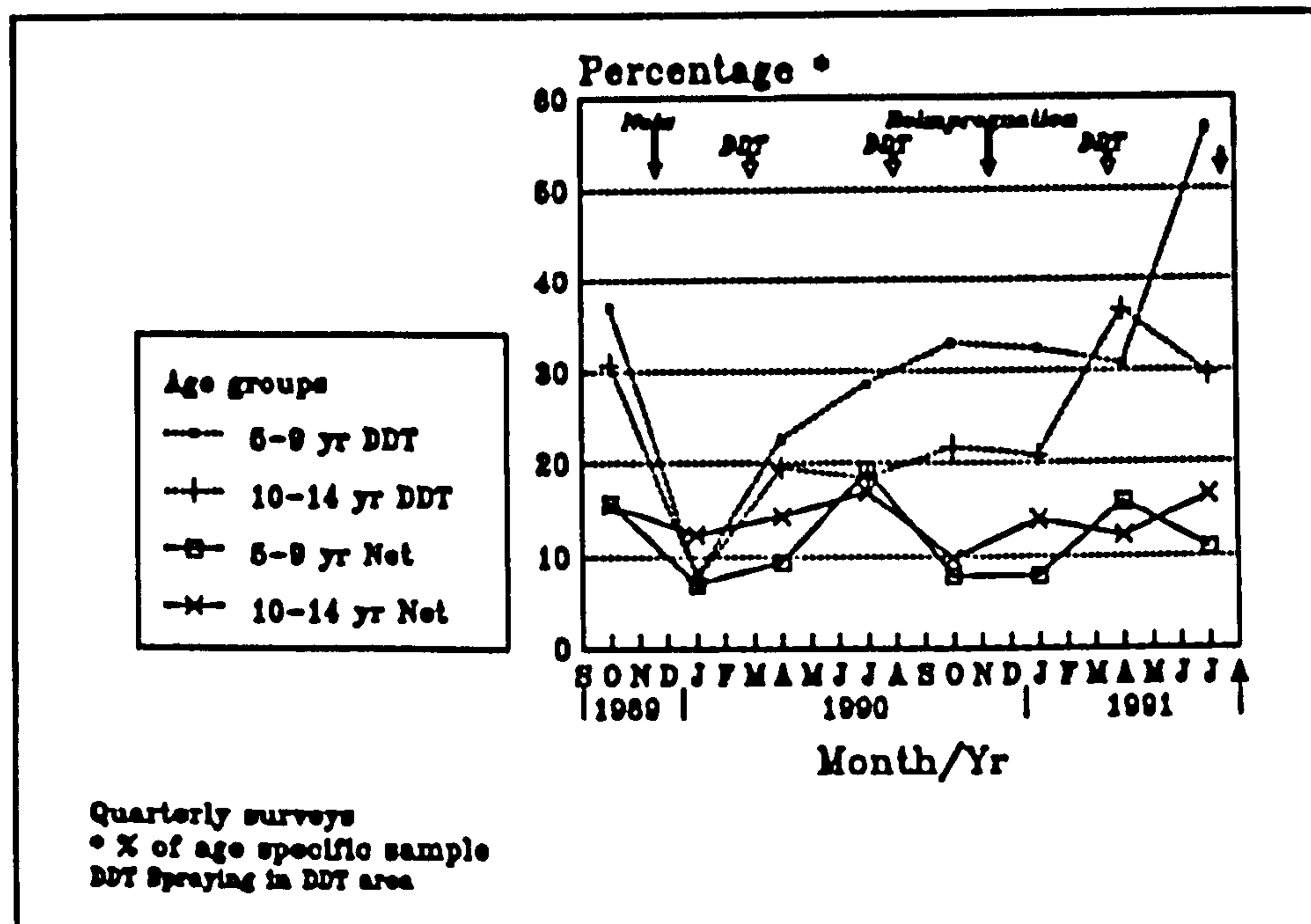
**Fig.16.** *Malaria prevalence in the under fives in the bednet and DDT trial areas, Isabel island.*



The prevalence in the five year old and over, 5-9 years and 10-14 years, is presented in Fig.17. The rates in both age groups in the DDT area were higher than the corresponding age groups in the bednet area. They dropped sharply in the second survey due to similar reasons discussed above, but, quickly rose to the same high levels at the beginning of the study. The rates in the bednet area dropped in the second survey and were low, but started to rise eight months after impregnated nets were introduced. They dropped again after the nets were re-impregnated and were maintained low, except a rise in the older age groups (10-14 year) in the last survey. This rise in the bednet area was due to P.vivax while the big increase in the DDT area was mainly due to P.falciparum.

The prevalence in the 15 years and older, and all ages combined is presented in Fig.18. The rates, with those in the DDT area initially higher than in the bednet area, all dropped in the

Fig.17. *Malaria prevalence in 5 to 14 year old in the bednet and DDT trial areas, Isabel island.*

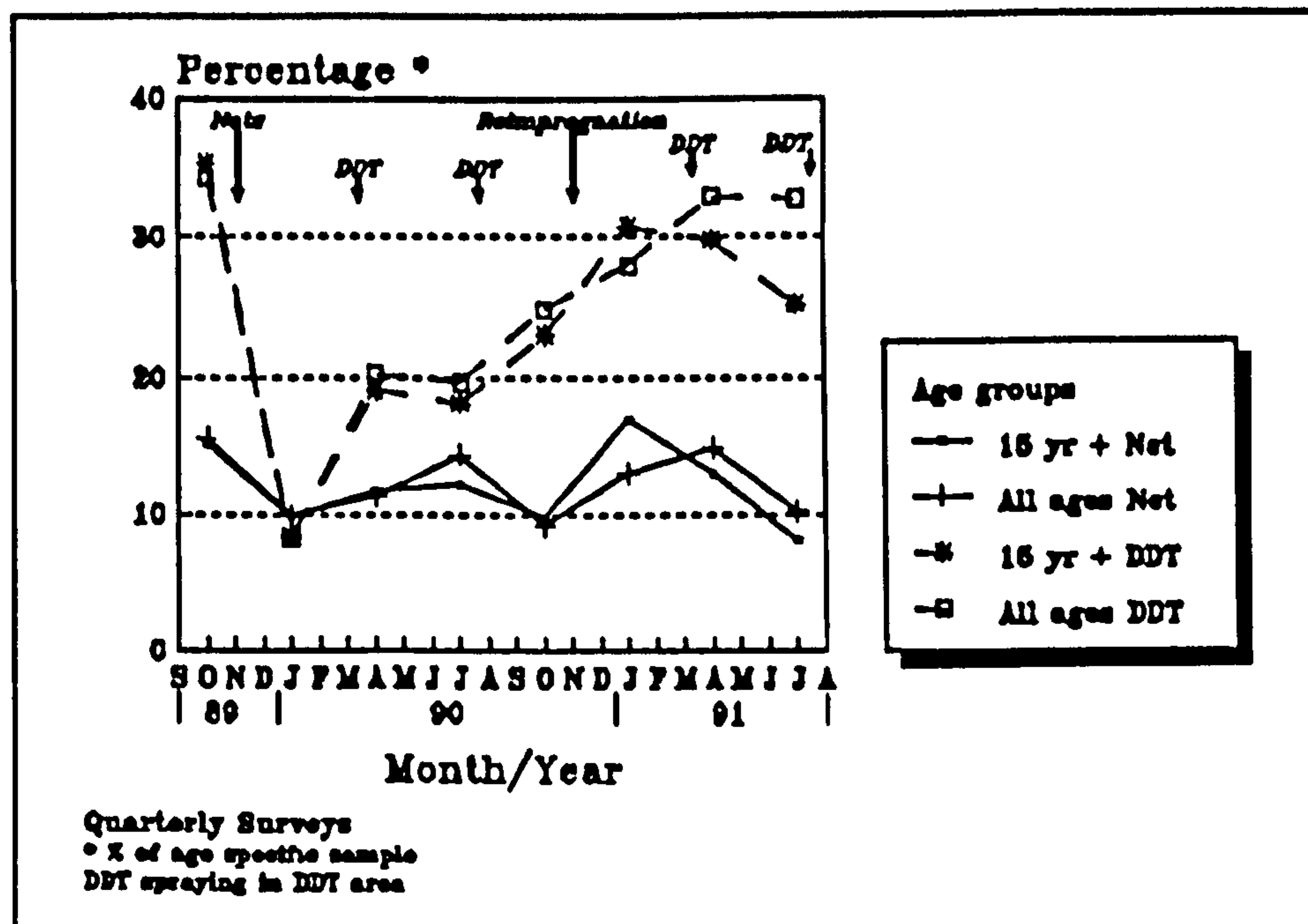


second survey. Those in the DDT area, irrespective of DDT spraying cycles, rose steadily to the initial level in all ages, and close to the initial level in the 15 years and older. In the bednet area the low rates were maintained, but with a rise eight months after nets were distributed. They dropped again after the nets were re-impregnated. The slightly rise in all ages in the last surveys was due to P.vivax cases mainly in older age groups.

The ratio between P.falciparum and P.vivax in the DDT area also started equally and remained about the same, 1 to 1.3 in the first survey, and by the last survey, it was 1.6 to 1. In the bednets area the ratio was 1.7 P.falciparum to one of P.vivax, at the start but by the end it came to one to 3. Therefore the reduction in the bednet area was mainly in younger age groups, and especially with P.falciparum. The majority of cases that occurred in the last surveys in the bednet group were in the older age groups, mostly of P.vivax (Table 11). It was likely that inoculation occurred outside bednets, particularly as this age group may tend to wander outdoors.



**Fig.18 Malaria prevalence in adults and all ages combined, in bednet and DDT areas, Isabel island.**



In the DDT area many cases still occurred in young age groups and were mainly of P.falciparum.

**Table 11. Age specific malaria prevalence (%) by the last survey, Isabel island trial area.**

AGE GROUP	DDT				BEDNET			
	Number	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	23	30.4	21.7	8.7	23	0	0	0
1-4	95	39.0	26.3	12.7	72	11.1	1.2	9.7
5-9	99	56.6	33.3	23.3	92	10.9	2.2	8.7
10-14	176	29.6	16.5	13.1	121	16.5	5.8	10.7
15plus	316	25.3	16.1	9.2	298	8.1	1.7	6.4
<b>TOTAL</b>	<b>709</b>	<b>32.7</b>	<b>20.2</b>	<b>12.6</b>	<b>606</b>	<b>10.2</b>	<b>2.5</b>	<b>7.8</b>

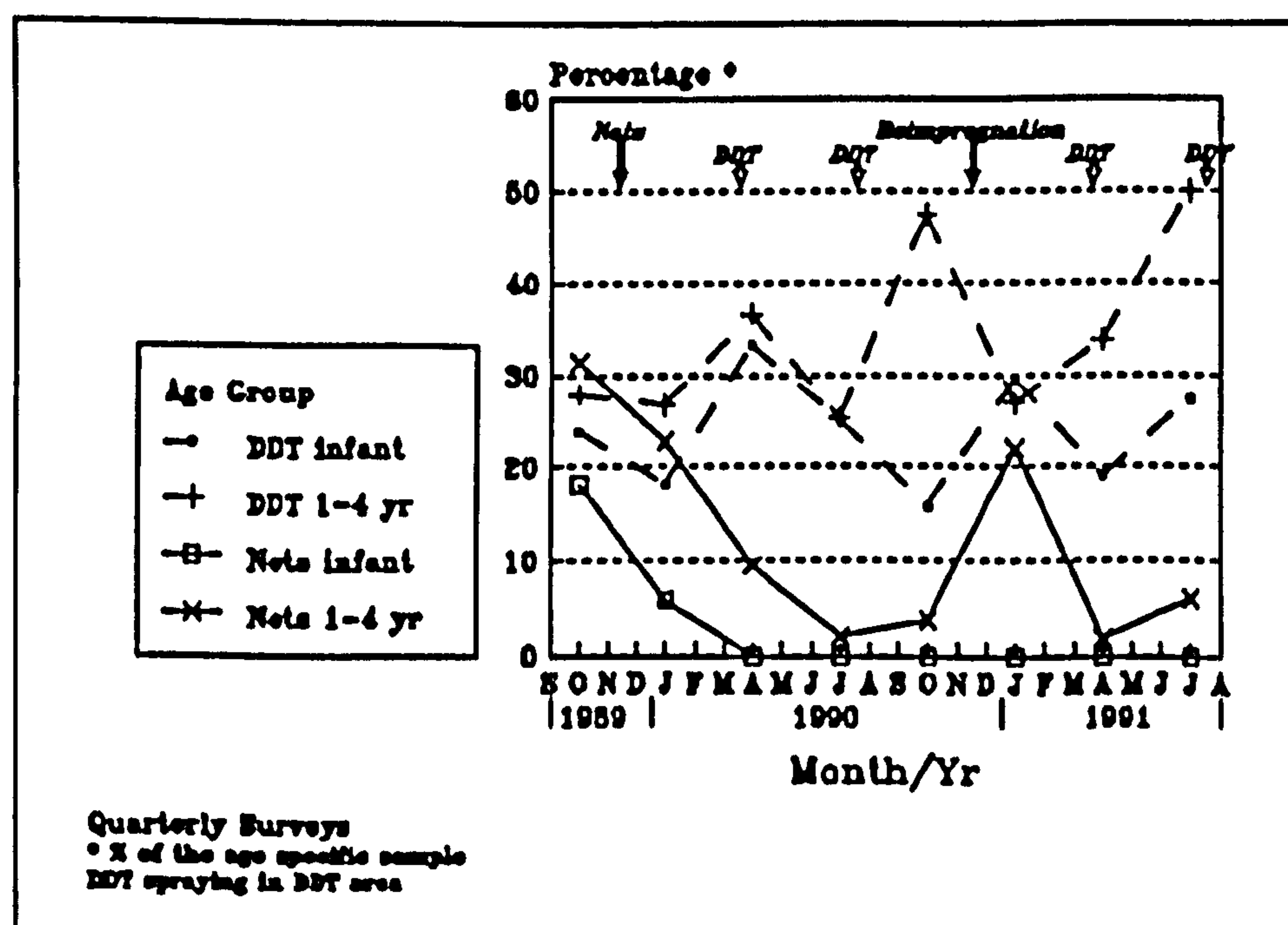
The communities in this study pair reasonably accepted DDT spraying in the past. At the end of the study there was no change in general prevalence with continuation with DDT residual spraying, even though there were fluctuations. While prevalence rates at the start were lower, further reduction was achieved with permethrin treated bednets. The trend in the DDT area

increased, particularly in the young age groups, and with P.falciparum. Thus impregnated bednets in Isabel study area reduced malaria prevalence, especially against P.falciparum and mainly in young age groups. Prevalence was maintained at a low level.

#### 4.2.2 Vella La Vella Island

The prevalence in the under fives, infants and 1-4 year old in the Vella La Vella trial area is presented in Fig.19. The rates in the DDT area, which were initially the same as in the corresponding age groups in the bednet area, did not show any sustained effect with DDT spraying. Even though there were variations which may have been an impact of DDT spraying,

**Fig.19 Malaria prevalence in the under fives in bednet and DDT trial areas, Vella La Vella island.**



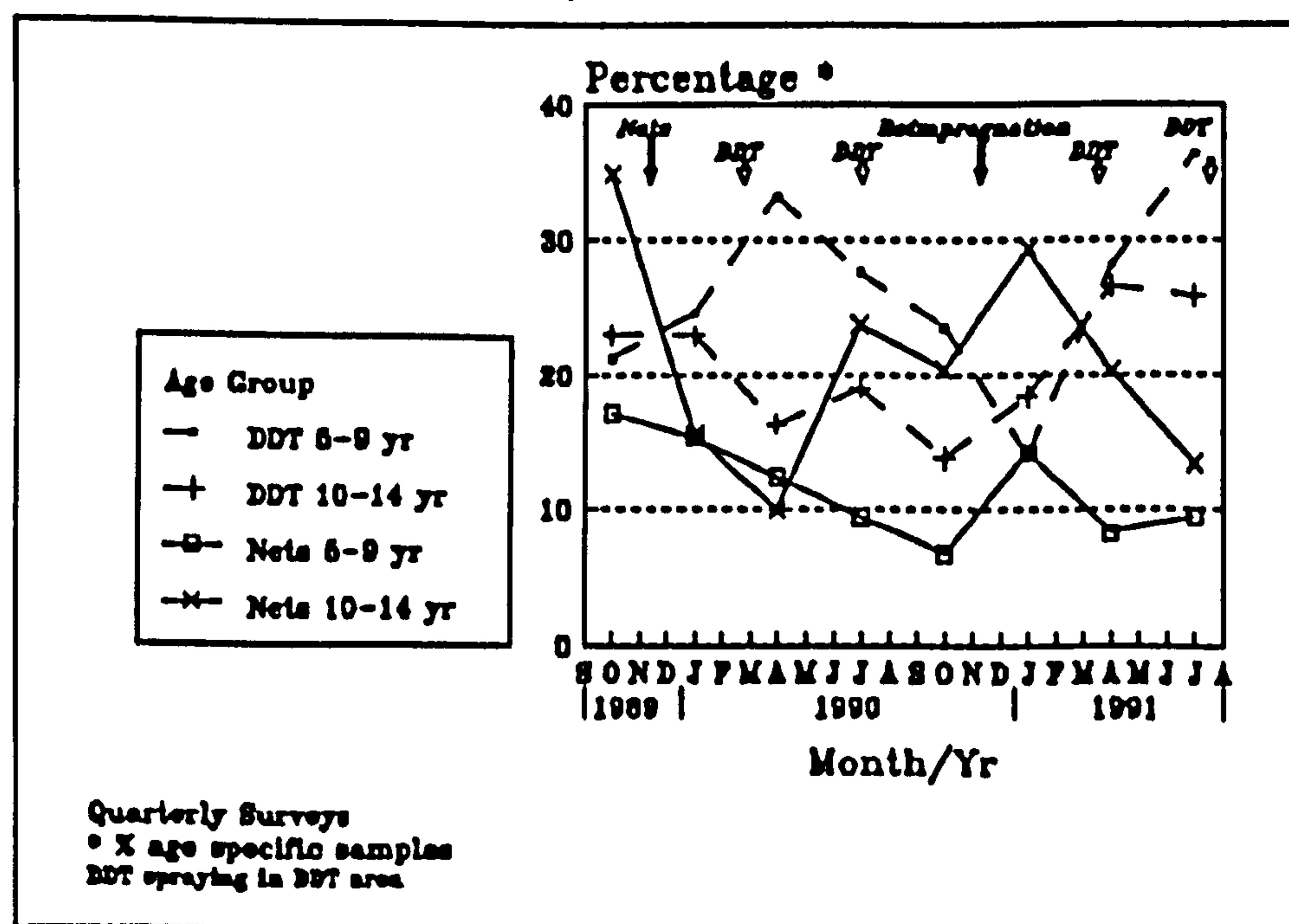
the rate in infants remained basically unchanged, and in the 1-4 year old it increased to almost 20% higher in the last survey. The rates in the bednet area declined and by the fifth month after the nets were distributed there were no more positives in infants. In the 1-4 year old the prevalence was kept low, except a spike in the sixth survey caused mainly by some positive P.vivax which was the species contributing to prevalence in bednets area. The rise in the DDT



area was mainly due to P.falciparum.

The prevalence in the 5-9 year, and 10-14 year old is presented in Fig.20. The rates in the bednet area dropped sharply with the introduction of impregnated bednets and in the 5-9 year old it declined further till a rise in the sixth survey. It declined again after re-impregnation. The rate in the 10-14 year age group was initially much higher than the corresponding age group in the DDT area, but dropped sharply, following a similar rise, after re-impregnation. This temporary rise in both age groups was due to P.vivax cases, the main species contributing to prevalence in the bednet area. In the DDT area the rate in the 5-9 year old rose to the highest level in the third survey, but dropped again before a steady rise, to the highest level, in the last

**Fig.20 Malaria prevalence in the 5 to 14 year old in the bednet and DDT trial areas, Vella La Vella island.**

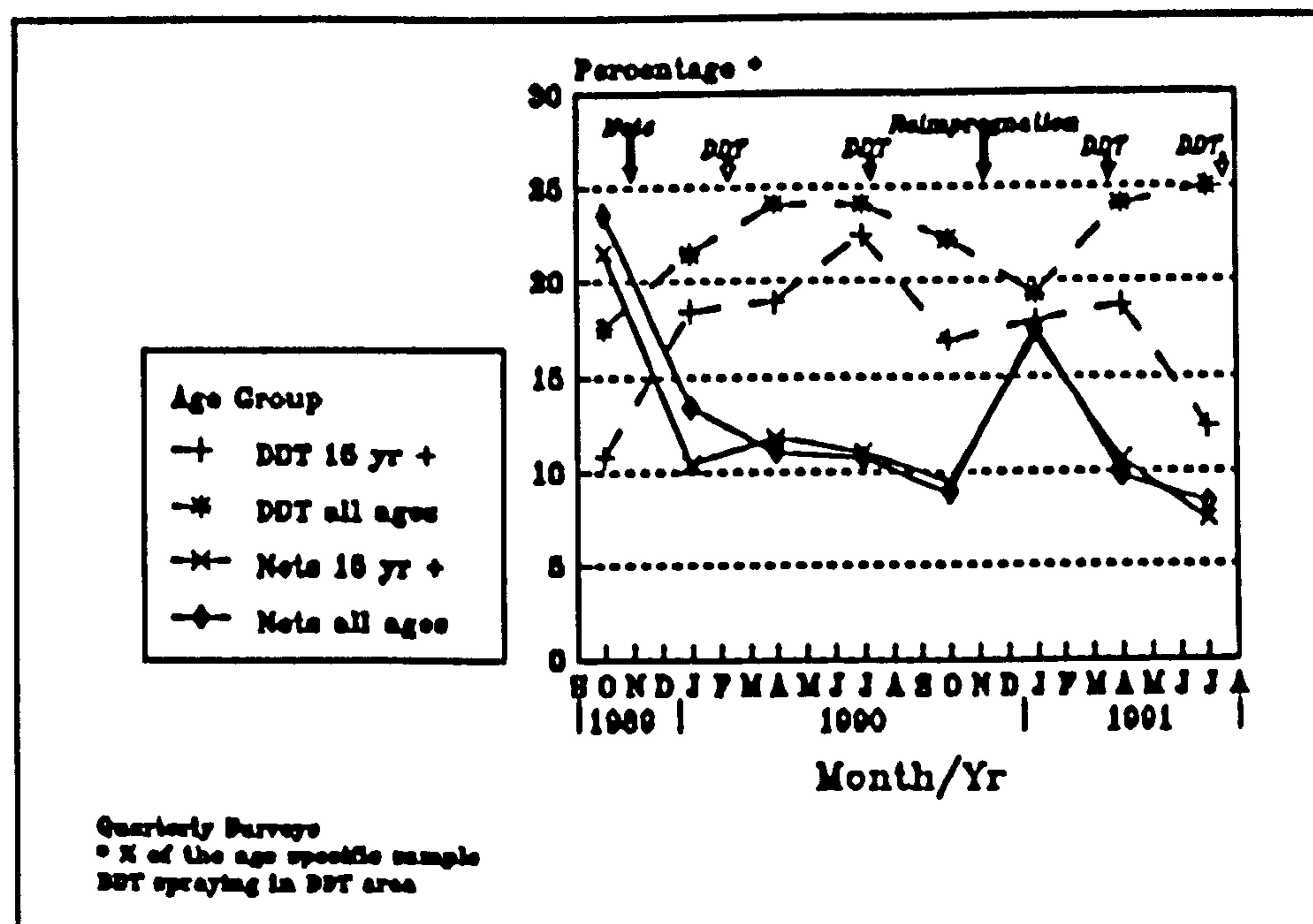


survey. The rate in the 10-14 year old rose steadily, even though there were variations. These variations might be due temporarily to DDT spraying, which at the end seemed to have no sustained impact. The increases in both age groups were mainly due to P.falciparum.

The prevalence in the 15 years and older, and all ages combined is presented in Fig.21. The rate in the DDT area of the 15 years and older started lower than its corresponding age



**Fig.21 Malaria prevalence in adults and all ages combined, in bednet and DDT areas, Vella La Vella trial area.**



group in the bednets area, but rose steadily till after the second cycle of DDT spraying when it declined slowly to almost the same initial level. The rate in the bednet area dropped sharply after nets were introduced and remained low till re-impregnation, when it rose slightly, but declined further to a low level of about 10%, mainly due to P.vivax infections. In all ages combined the drop after introduction of bednets was maintained at around 10%, except for a temporary increase in the sixth survey, mainly some P.vivax cases in all ages (except infants). The prevalence in all ages in the DDT area started lower but rose steadily, unaffected by DDT spraying cycles, to the highest level in the last survey and with P.falciparum as the main species.

In general the prevalence at the beginning was higher in the bednet area than in the DDT area. There were fluctuations but it increased in the DDT area, especially with P.falciparum, which became two to one of P.vivax. In the bednet area the large number of cases at the start were in the younger age groups, and mainly with P.falciparum (ratio 1.4 to 1 P.vivax). Even though there were fluctuations in trend, there was a general decline, much more marked in the younger age groups, and especially with P.falciparum. The ratio became one P.falciparum to

three P.vivax.

Most cases that occurred in the bednets area in the last survey were in older age groups, and mainly of P.vivax malaria (Table 12). It was likely that older people remained outside bednets, particularly outdoors, and were infected by young adult vectors with infective P.vivax. The majority of cases in the DDT area were in the younger age groups, mainly P.falciparum. It seemed that impregnated bednets maintained the prevalence of malaria at a low level, except in infants and under fives, they did not reduce it less than about 10%, with some persistence P.vivax transmission.

**Table 12.** Age specific prevalence (%) by the last survey, Vella la Vella island trial area.

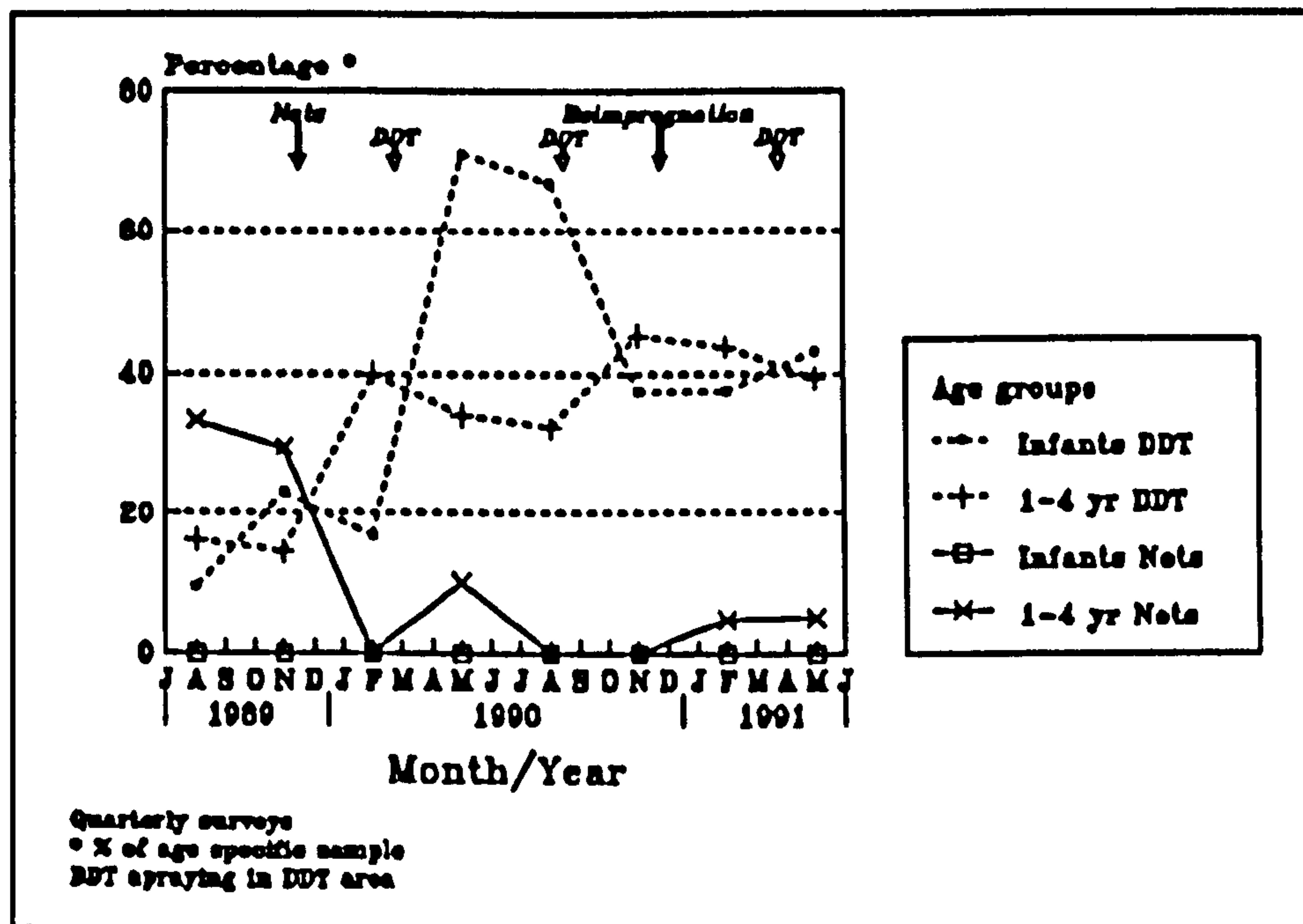
AGE GROUP	DDT				BEDNET			
	Number	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	22	27.3	18.2	9.1	9	0	0	0
1-4	60	50.0	35.0	15	49	6.1	0	6.1
5-9	63	36.5	19.0	17.5	63	9.5	3.2	6.3
10-14	58	25.9	15.6	10.3	52	13.5	3.9	9.6
15 plus	185	12.4	7.0	5.4	163	7.4	2.5	4.9
TOTAL	388	25.0	15.2	9.8	336	8.3	2.4	5.9

#### 4.2.3 Vona Vona/Kolobangara Islands

In the third trial, Vona Vona islands was the DDT area and Kolobangara the permethrin impregnated bednet area. The prevalence in infants and 1-4 year old are presented in Fig.22. The rates in the DDT area, initially lower than those of corresponding age groups in bednets area, steadily rose to levels 20% higher than the initial levels at the end of the study. There were maximum rates in infants in the fourth and fifth surveys, due to positive cases as well as small samples. DDT spraying cycles had no impact on the prevalence. In the bednet area there was no



**Fig.22 Malaria prevalence in the under fives in Vona Vona (DDT) and Kolobangara (bednet) trial area.**



positive case detected in infants throughout the study. But in the 1-4 year old there was a dramatic drop after impregnated bed nets were issued and the low level was maintained. Few cases were detected in the fourth, seventh and eighth surveys. The reduction in the bednet area, and increases in the DDT area, were mainly with P.falciparum.

The prevalence in the 5-9 year, and 10-14 year old are presented in Fig.23. The rate in the 5-9 year old in the DDT area, initially lower than those in the bednet area of the same age groups, rose rapidly, especially with P.falciparum, but declined slightly in the last two surveys, to a level 10% higher than the initial values. The variation was unaffected by DDT spraying cycles. The rate in the 10-14 year old in the same intervention area seemed to decline temporarily after each DDT spraying cycles, except the third cycle, when it rose to more than 10% of initial level. These variations even though there may have been some unsustained positive effect of DDT spraying, could also be due to other factors, as by the end there was a general increase, especially with P.falciparum.

The rates in the bednet area dropped sharply after nets were introduced and remained low,



especially in the 5-9 year old. Then, particularly in the 10-14 year old, prevalence started to increase eight months after impregnated bednets were introduced, but declined again after the

**Fig.23 Malaria prevalence in the 5 to 14 year old in Vona Vona (DDT) and Kolobangara (bednet) trial area.**

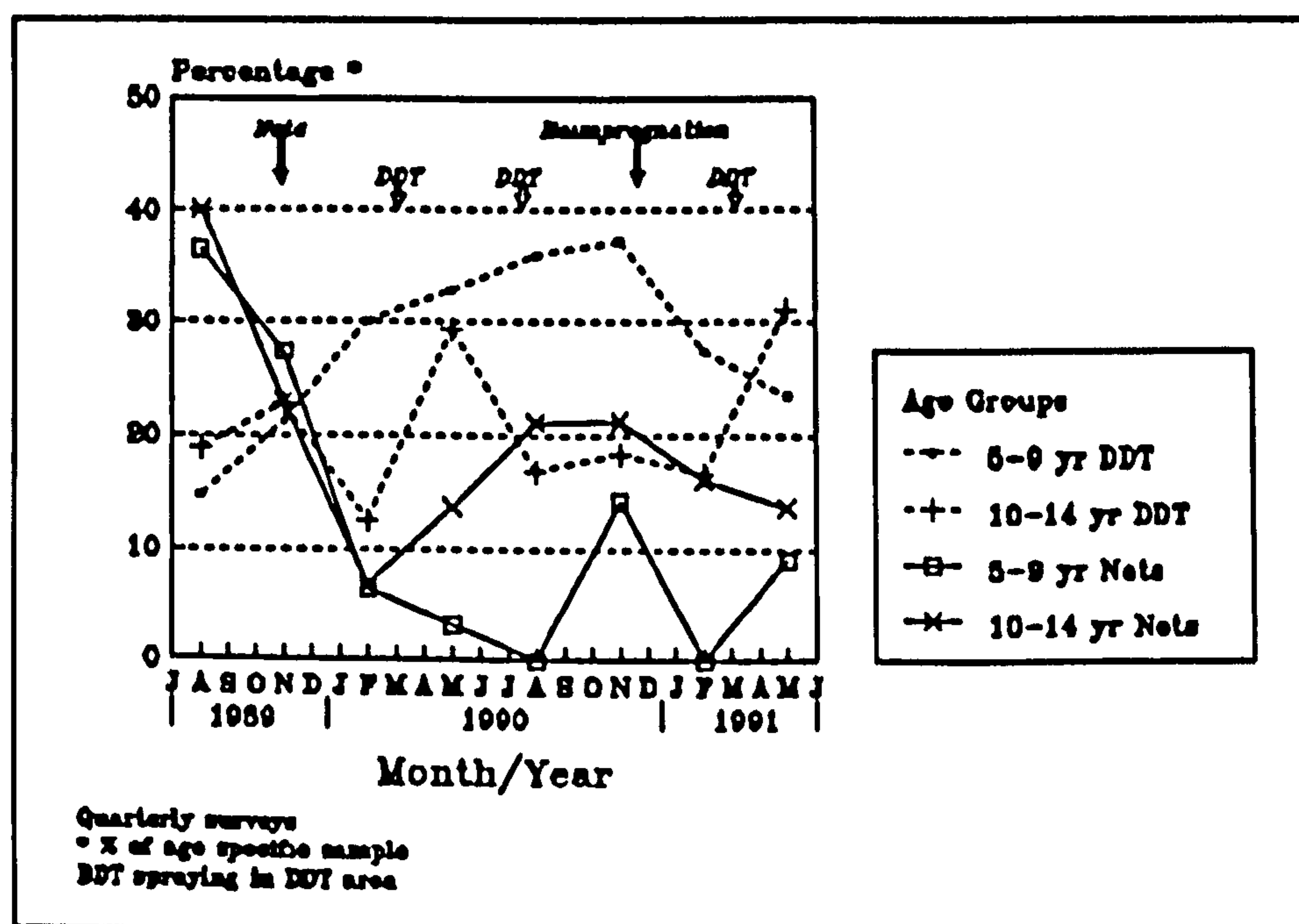
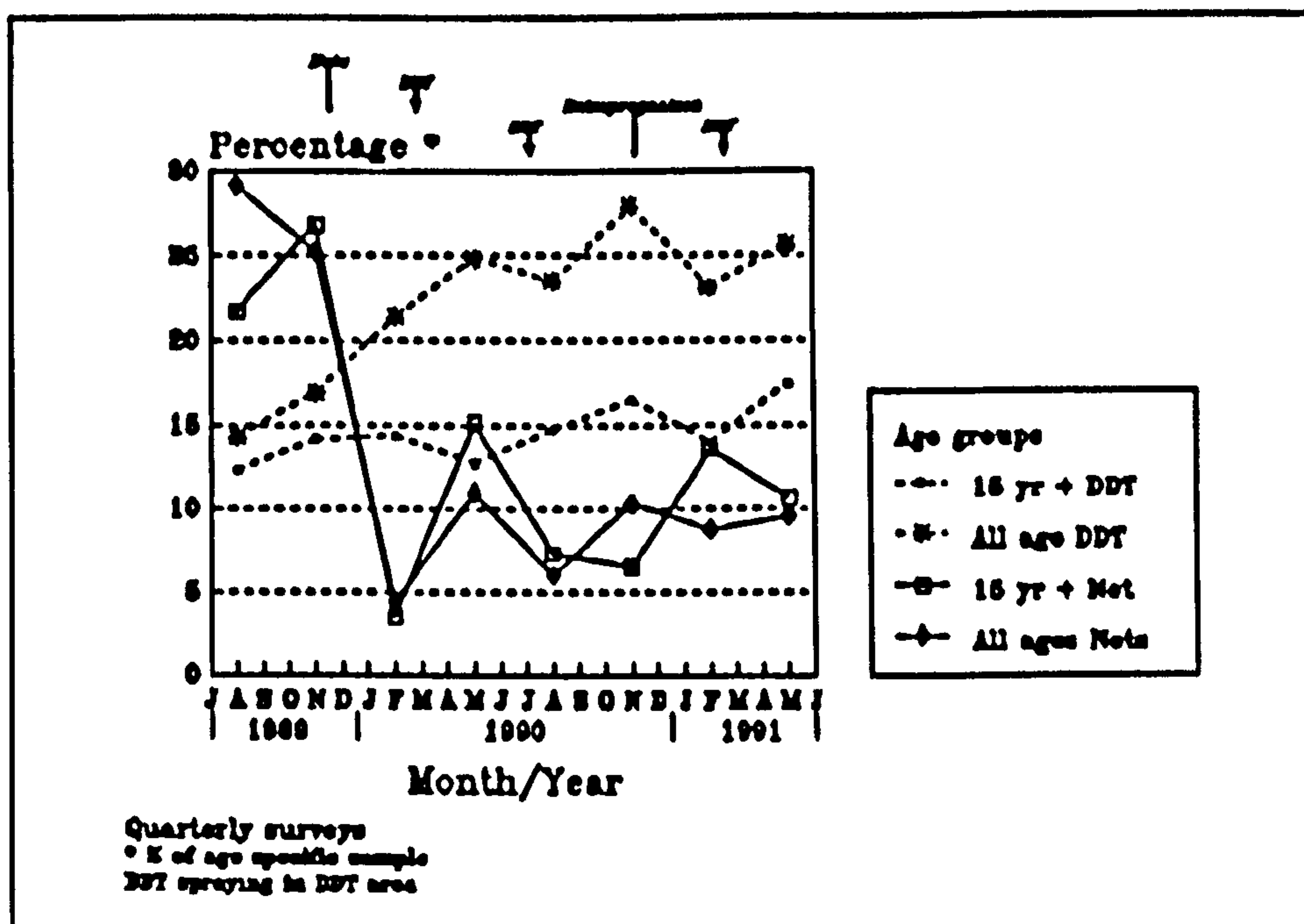


Fig.24 Malaria prevalence in adults and all ages combined in Vona Vona (DDT) and Kolobangara (bednet) trial area.



The P.falciparum to P.vivax ratio in all ages at the beginning was 1.1 to 1 in the DDT area, and 1.9 to 1 in the bednet area. By the end it was, 1.9 to 1 in the DDT area, but reversed slightly to 1 to 1.3 in the bednet area. The reduction in the bednet area was especially marked in the younger age groups and with P.falciparum. In terms of numbers of positive cases in the last survey, the majority in the bednet area were in the older age group, especially with P.vivax. In the DDT area the species was still predominantly P.falciparum, particularly in the younger age groups (Table 13). Compliance with DDT was initially high in the DDT area prior to the study, with almost similar parasite ratios. But after two years the prevalence increased including infections in infants, even though the samples in infants were small.

In the bednet area, there was an epidemic of malaria nine months prior to the study. The remedial measures taken then included an episode of ULV spraying in the communities, but the prevalence rates were still higher than in the DDT area at the start. After introduction of impregnated bednets the rates declined in all ages even though there were fluctuations in the older age groups. In this pair permethrin impregnated bednets effectively reduced prevalence of



malaria especially with P.falciparum in the younger age group.

**Table 13.** Age specific malaria prevalence (%) by the last survey Vona Vona/Kolobangara Islands trial area.

AGE GROUP	DDT				BEDNET			
	Number	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	7	42.9	28.6	14.3	5	0	0	0
1-4	56	39.3	25.0	14.3	20	5.0	0	5.0
5-9	68	23.5	16.2	7.4	33	9.1	0	9.1
10-14	45	31.1	20.0	11.1	29	13.8	6.9	6.9
15 plus	121	17.4	11.6	5.8	60	10.0	5.0	5.0
TOTAL	297	25.6	16.8	8.7	147	9.5	4.1	5.4

There were increases in the DDT area especially with P.falciparum and mainly in younger age groups.

#### 4.2.4 Florida Islands.

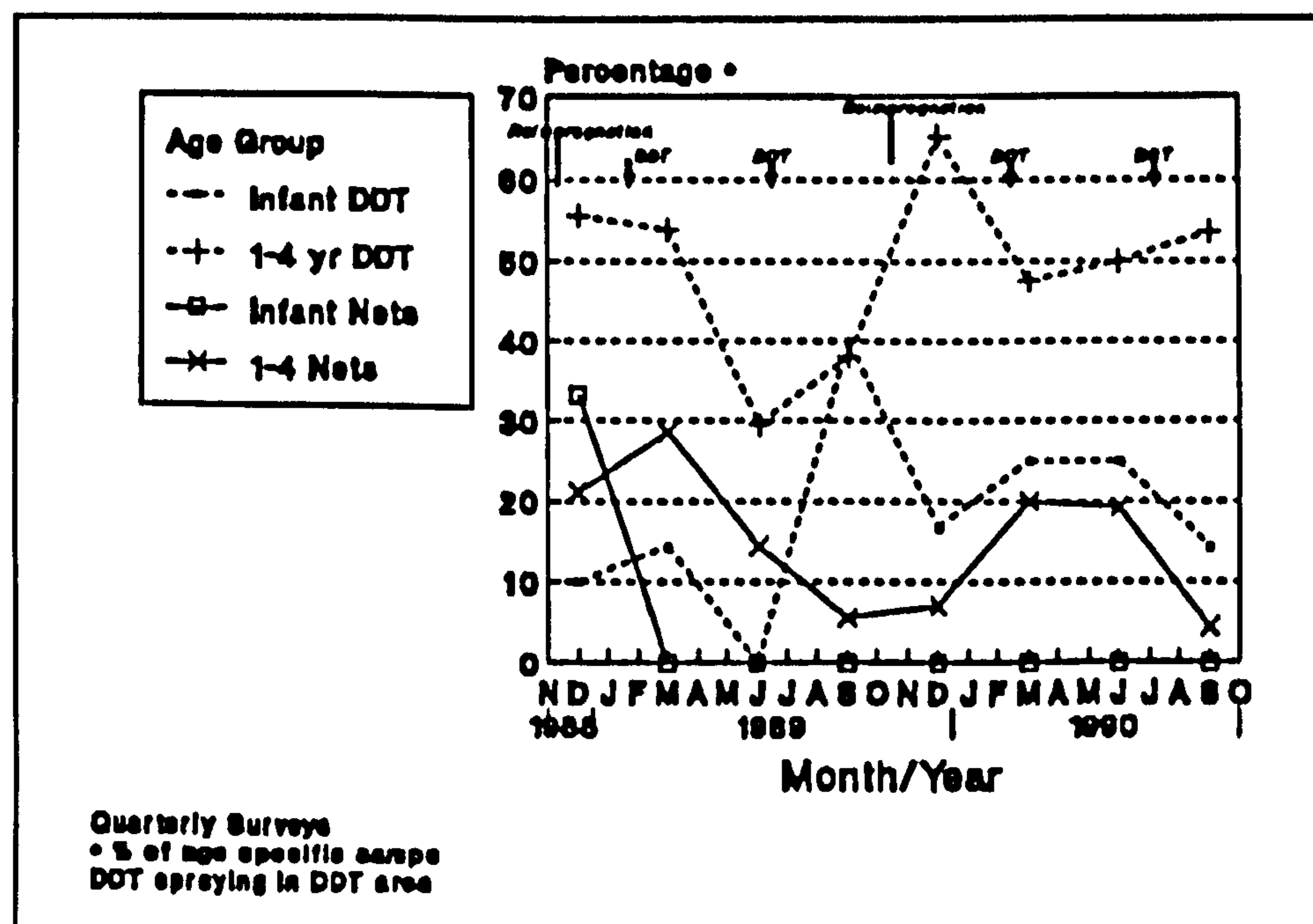
In the Florida Islands trial area the prevalence in infants and 1-4 year old are presented in Fig.25. The rates in infants in the bednet area immediately declined to zero, and was maintained as such throughout. In the DDT area the rate in infants, initially lower than that in bednets by over 20%, declined to zero after the first cycle of DDT, but quickly rose, and was maintained at a prevalence of 20%. The prevalence in the 1-4 year old in the DDT area, initially very high at 55%, dropped after the first cycle of DDT spraying. But it quickly rose after the second cycle, and despite continued DDT spraying, was maintained at prevalence of 50%. The rate in the bednet area in the 1-4 year old declined to less than 10%, but rose slightly with P.vivax cases and did not decline till well after the nets were re-impregnated. P.vivax was the main species contributing to the residual transmission in the bednets area, and some may have



been infected before the nets were reimpregnated.

The prevalence in the 5-9 year, and 10-14 year old are presented in Fig.26. There were variations in these age groups in the DDT area. The rate in the 5-9 year in the DDT area, which were highest at 66%, declined immediately with DDT spraying cycles. But after the second cycle the rate increased and fluctuated, with a final rate of 35% in both age groups. The third and

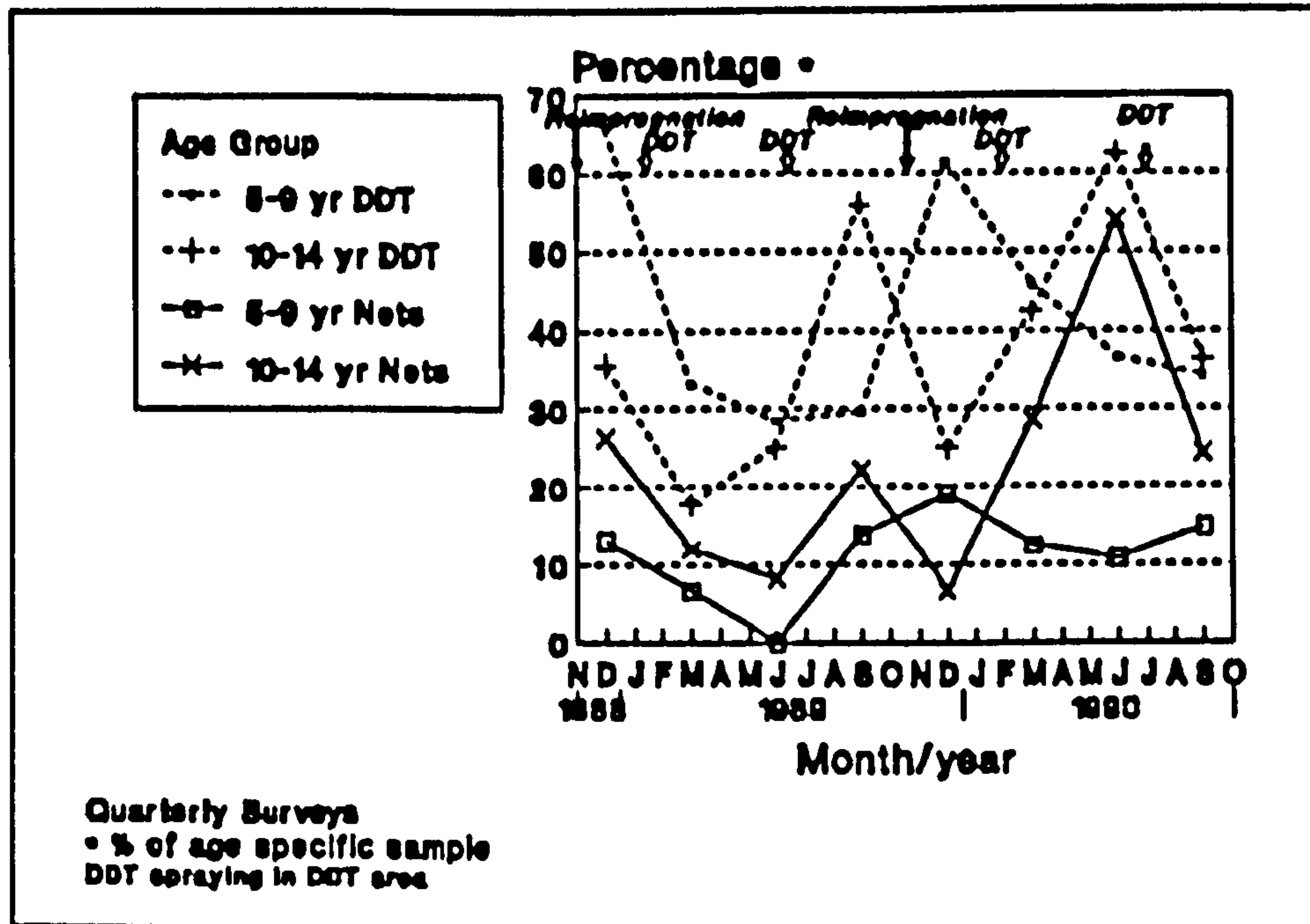
**Fig.25 Malaria prevalence in the under fives in the bednet and DDT trial areas, Florida islands.**



fourth cycles of DDT spraying might have affected prevalence in the 5-9 year age group, while the fourth the 10-14 year old. These rises were mainly with P.falciparum. The final rates were lower than their initial values but still higher than those of the same age groups in the bednet area. The rates in the bednet area declined slightly after re-impregnation of nets, and were maintained at around 10%. There were some slight increase eight months after the nets were reimpregnated again, but declined again after the nets were reimpregnated, except in the 10-14 year old with an increase due to an outbreak of P.vivax. P.vivax was the species maintaining some transmission in the bednet area in these age groups.

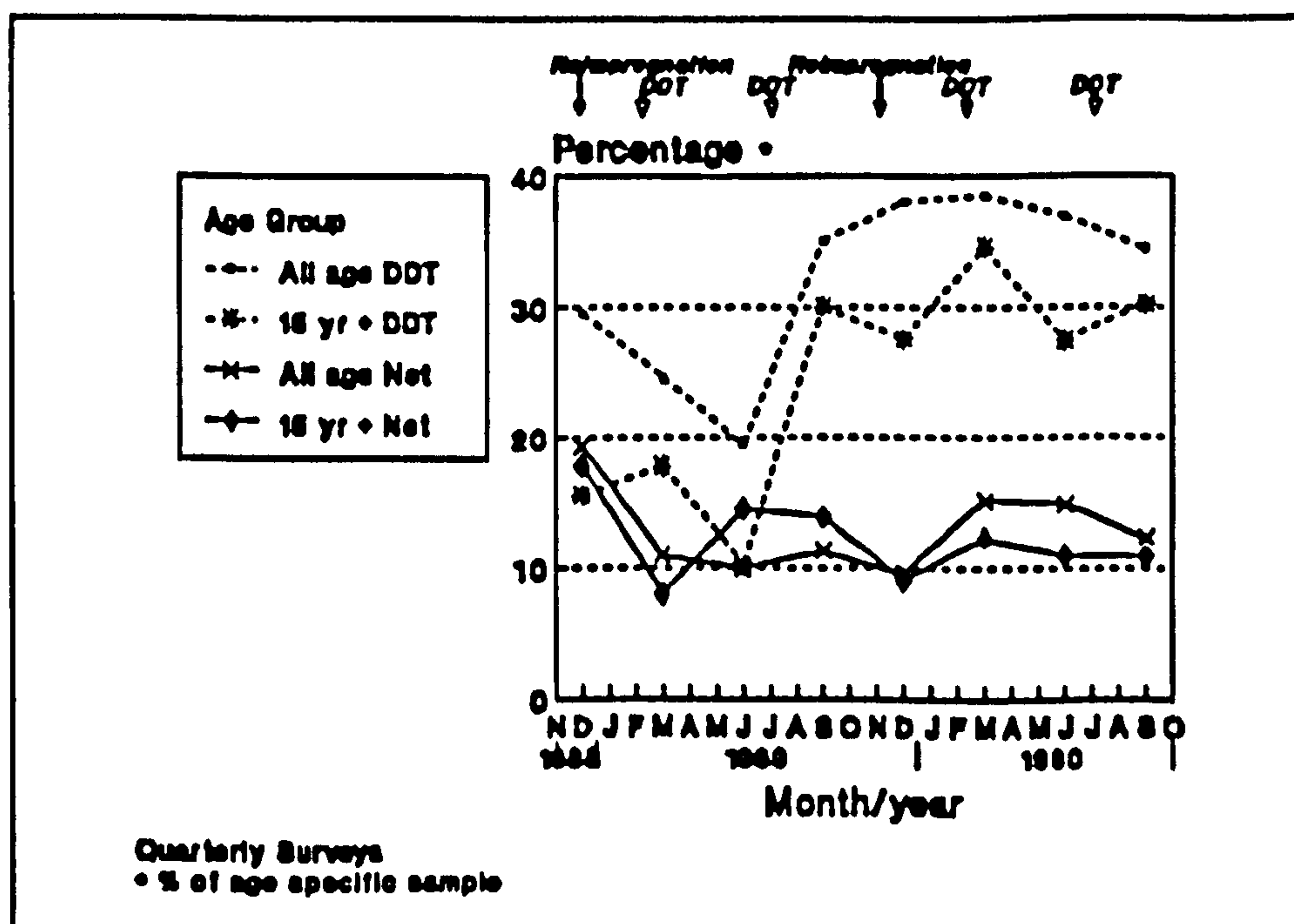
The prevalence in the 15 years and older, and all ages combined are presented in Fig.27.

**Fig.26 Malaria prevalence in the 5 to 14 year old, in the bednet and DDT trial areas, Florida islands.**



In the 15 years and older, the rate, initially at the same level as that in the bednet area in the same age group, declined briefly after the first cycle. Subsequently it rose quickly and was maintained at a prevalence of 30%. Both *P.falciparum* and *P.vivax* contributed to the high

**Fig.27 Malaria prevalence in adults and all ages combined in the bednet and DDT trial areas, Florida island.**



prevalence in the DDT area in this age group. In the bednet area the rate declined and was



maintained at 10%. The slight rise after bednets were reimpregnated was mainly of P.vivax. The rate in all ages in the DDT area declined immediately after the first cycle, but rose after the second cycle of DDT spraying and was maintained at 35%, 20% higher than in the bednet area. The rate in all ages combined in the bednet area declined and was maintained at 10%. The slight increase immediately after bednets were reimpregnated was due to P.vivax outbreaks in older age groups, especially in the 10-14 year old.

Even though there were fluctuations there was not much change in the overall trend in the DDT area. It increased especially in the younger age groups in the last surveys, especially with P.falciparum. The species ratio in the DDT area increased from being equal at the beginning, to 2.7 P.falciparum to 1 P.vivax, at the end.

In the bednet area, even though the rates were lower at the start they dropped further to around 10%. The biggest decline was in the younger age groups, and especially with P.falciparum. The majority of positive cases in the last surveys were mostly in the older age groups, and mainly with P.vivax (Table 14).

It was the older age groups therefore that may have become infected outside bednets, or outdoors earlier in the evening, by A.farauti infective with P.vivax.

**Table 14. Age specific malaria prevalence (%) by the last survey, Florida Is. trial area.**

AGE GROUP	DDT				BEDNET			
	Number	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	7	14.3	14.3	0	4	0	0	0
1-4	28	53.6	42.9	10.7	23	4.4	4.4	0
5-9	35	34.3	22.9	11.4	27	14.8	7.4	7.4
10-14	22	36.4	22.7	13.6	17	29.4	5.9	23.5
15 plus	90	30.3	22.2	8.1	92	10.9	3.3	7.6
TOTAL	191	34.6	25.1	9.4	163	12.3	4.3	8.0

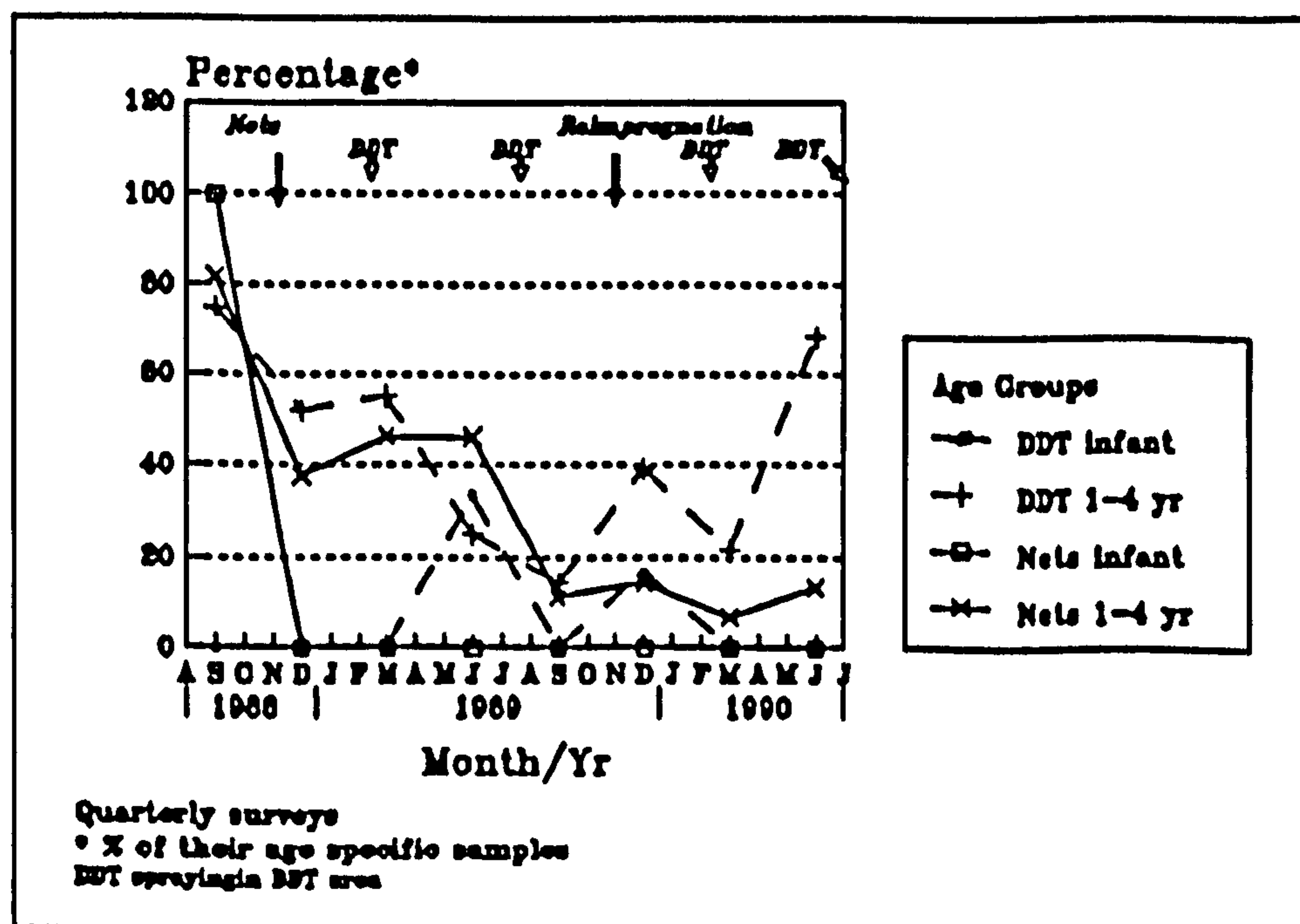


#### 4.2.5 Malaita Island

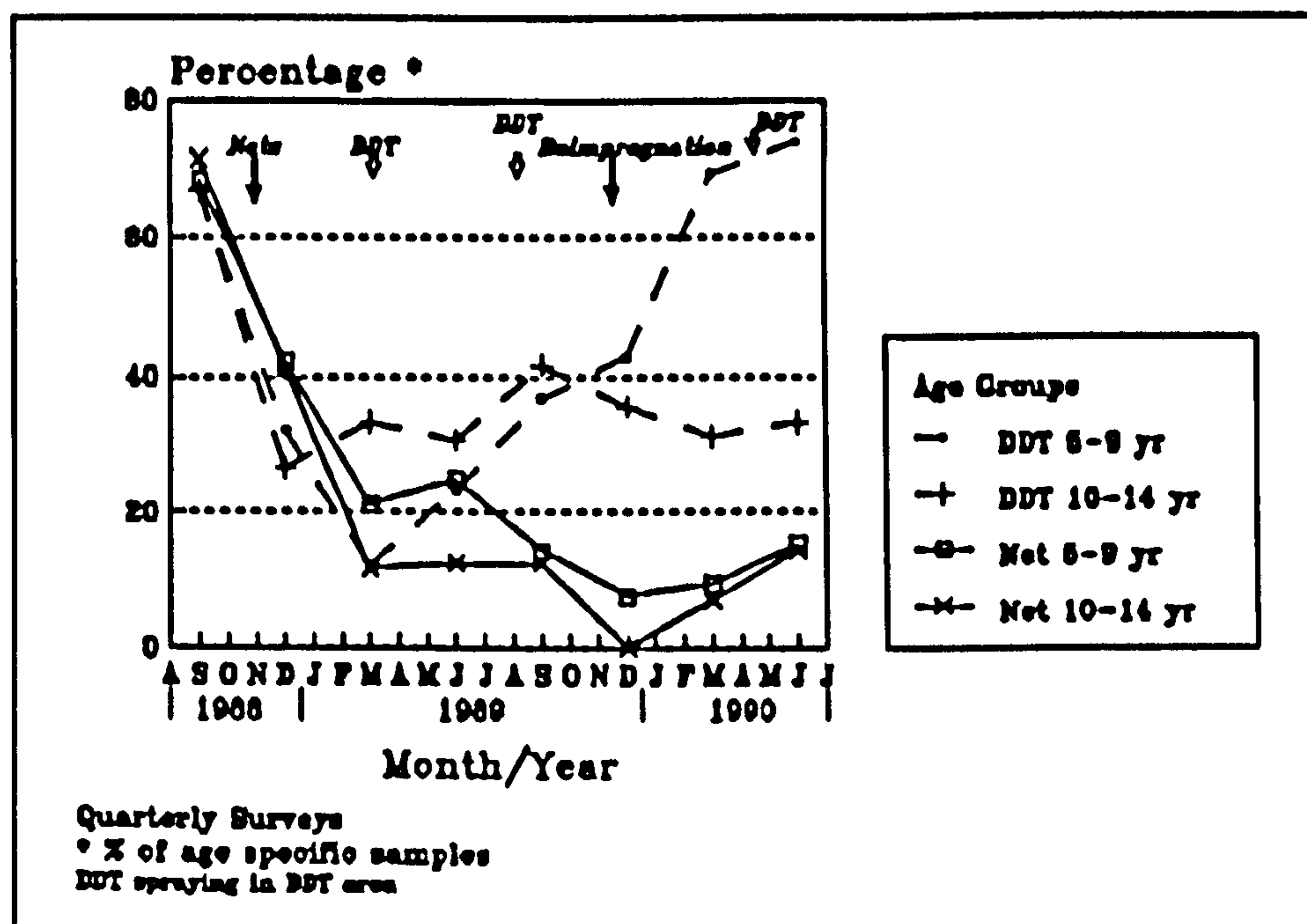
The prevalence in the under fives in the Malaita trial area is presented in Fig.28. The rate in infants in the bednet area dropped immediately to zero after bednets were introduced and remained so throughout the study. In the DDT area cases were only diagnosed in the fourth and sixth surveys. Both interventions provided effective protection in infants even though the samples in this age group were small. In the 1-4 year old the rates in both interventions, initially the same, declined. These reductions were especially with *P.falciparum*. It was reduced further with re-impregnation and maintained low at around 10% in the bednet area. In the DDT area it rose again after the second DDT spraying cycle, despite continued spraying, to a level 10% lower than the initial, but over 30% higher than that of the same age group in the bednet area. The apparent positive effect of DDT was not maintained.

The prevalence in the 5-9 year and 10-14 year old are as in Fig.29. All initial rates in both age groups were the same and all declined with interventions. The rate in the 5-9 year old in the DDT area declined further but quickly rose by the fourth survey, to over 70% in the last

Fig.28 *Malaria prevalence in the under fives in the bednet and DDT trial areas, Malaita island.*



**Fig.29 Malaria prevalence in the 5 to 14 year old in the bednet and DDT trial areas, Malaita island.**

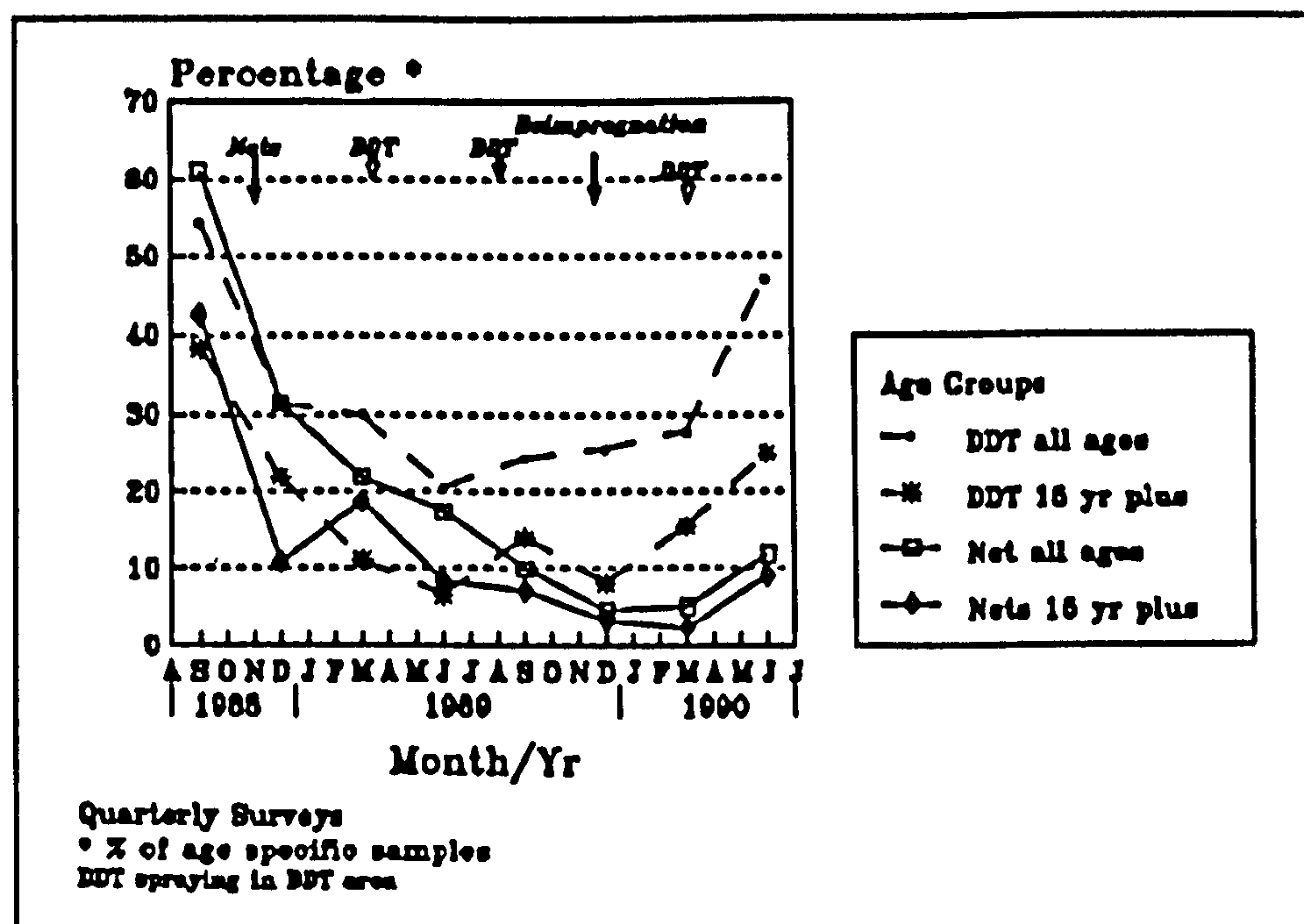


survey. The rate in the 10-14 year old in the DDT area increased early, maintained at around 30%, unaffected by DDT spraying. These increases in the DDT area were mainly with P.falciparum. In the bednets area the rates in both age groups declined further after the nets were reimpregnated. Prevalence was maintained at lower than 15%, mainly with P.vivax infections, in both age groups.

The prevalence in the 15 year and older and all ages combined are as in Fig.30. The rates in the 15 years and older, initially the same at 40% in both intervention areas, declined with both interventions. The rate in the DDT area only rose again in the last two surveys despite continued DDT spraying, to a level 15% lower than the initial value, but 15% higher than the same age group in the bednet area. The rate in the bednet area declined further after re-impregnation and was maintained at about 10%, mainly with few P.vivax cases. The rates with all ages combined in both intervention areas declined initially, but in the DDT area rose again gradually after the second DDT spraying cycle, up to 10% below the initial level in the last survey. In the bednet area prevalence was maintained at the low 10% level.



Fig.30 *Malaria prevalence in adults and all ages combined in the bednet and DDT trial areas, Malaita island.*



On the trial area in Malaita both intervention areas at the start had similar rates of infection especially with P.falciparum, and mainly in the younger age groups. The P.falciparum to P.vivax ratio at the start was, 19 to 1 in the DDT area, and 2.4 to one in the bednets area. At the end the respective ratios were 10.1 to 1 in the DDT area and 1 to 1.4 in the bednet area.

A.punctulatus was the main vector in this trial area where no vector control measures had been applied for several years. This vector quickly disappeared especially with permethrin impregnated bednets. There was a positive effect of DDT in reducing the prevalence of infections to a lower level, but on the second year cases were on the increase in the younger age group and especially with P.falciparum. The initial positive effect of DDT was not maintained. This may be due either to poor DDT spray coverage, or by A.farauti becoming the predominant vector with the decline of A.punctulatus. The prevalence rates were high in the bednet area initially, mainly with P.falciparum in the younger age groups. But the decline was dramatic after introduction of treated bednets in all ages, especially in the younger age groups. The rates were also maintained in all ages at about 10%. The majority of the cases that occurred were in the



**Table 15. Age specific malaria prevalence (%) by the last survey, Malaita Island Trial area.**

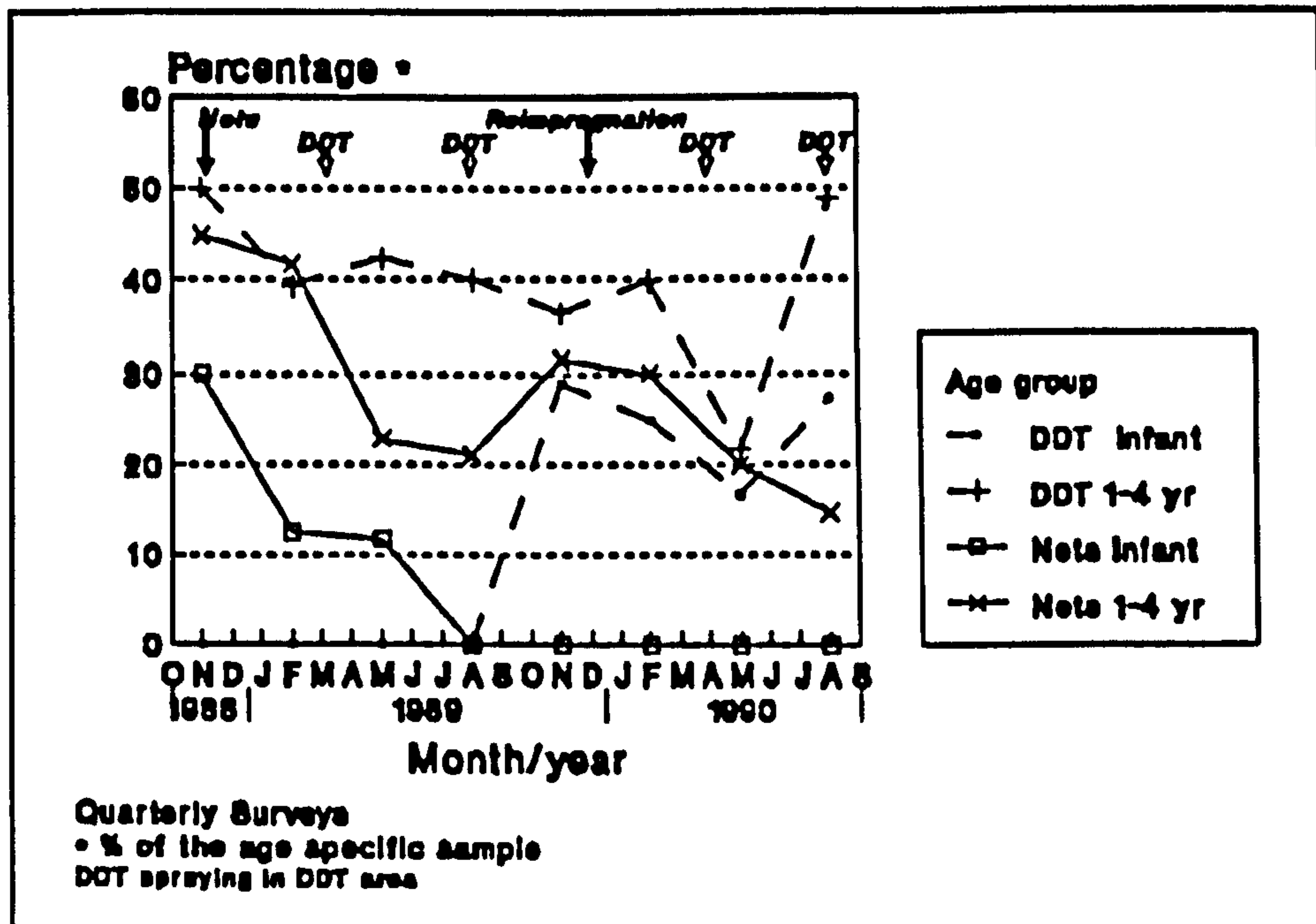
AGE GROUP	DDT				BEDNET			
	Total	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	3	0	0	0	3	0	0	0
1-4	22	68.2	68.2	0	15	13.3	0	13.3
5-9	23	73.9	56.5	17.4	26	15.4	3.8	11.6
10-14	18	33.3	33.3	0	14	14.3	7.1	7.1
15 plus	32	25.0	25.0	0	44	9.1	6.8	2.3
TOTAL	98	46.9	42.8	4.1	102	11.8	4.9	6.9

older age groups, and mainly of P.vivax malaria (Table 15). It may be that as bednets were used the reduction of the main vector was high. The older age groups therefore might have tended to wander outside the nets and be infected outdoors. There were some A.farauti which might have been mostly biting outdoor transmitting mainly P.vivax. In the DDT area, most cases occurred in the younger age groups, and mainly with P.falciparum.

#### 4.2.6 Guadalcanal Island One.

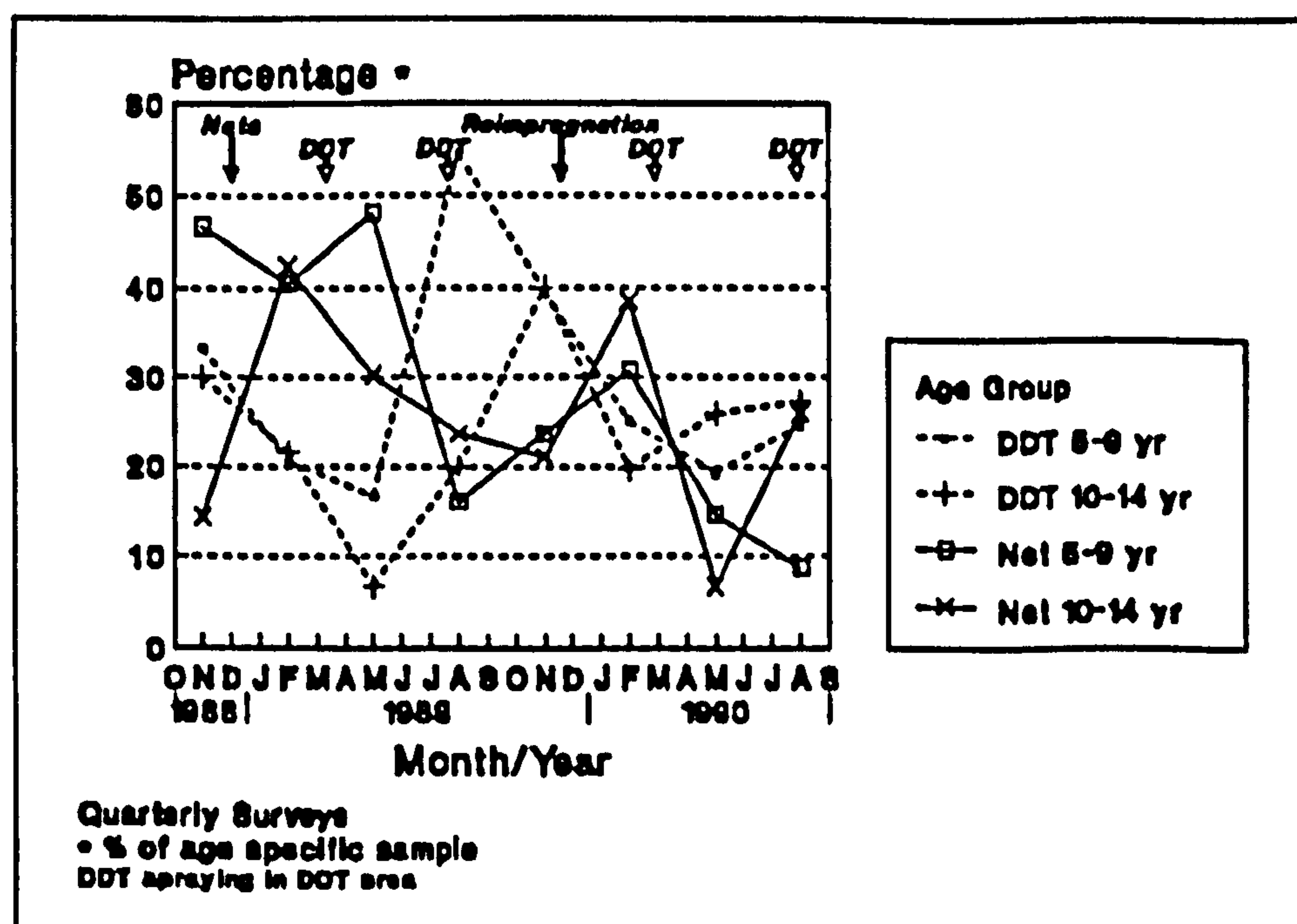
The prevalence in the first Guadalcanal trial area in infants and 1-4 year old are shown in Fig.31. There were no samples in infants in the first three surveys in the DDT area. The rate was between 20% to 30% in the last three surveys, even though samples were small. The rate in infants in the bednet group declined after impregnated bednets were introduced, to zero since the fourth survey. The rate in the 1-4 year old in the DDT area declined gradually from the initial level of 50%, till the last survey when it rose again to almost the same level. This increase was mainly with P.falciparum. The rate in the 1-4 year old in the bednet area, initially just below 40%, declined to 20% before starting to rise. It declined after the nets were reimpregnated to less than 15%, mainly with P.vivax. The main reduction was more marked with P.falciparum.

**Fig.31 Malaria prevalence in the under fives in the bednet and DDT trial areas, Guadalcanal One.**



The prevalence in the 5-9 year and 10-14 year old are presented in Fig.32. The rate in the 5-9 year old in the DDT area, initially lower than that of same age group in the bednet area, declined with DDT spraying, but rose to a peak in the fourth survey. It then declined again to

**Fig.32 Malaria prevalence in the 5 to 14 years old in the bednet and DDT trial areas, Guadalcanal One.**



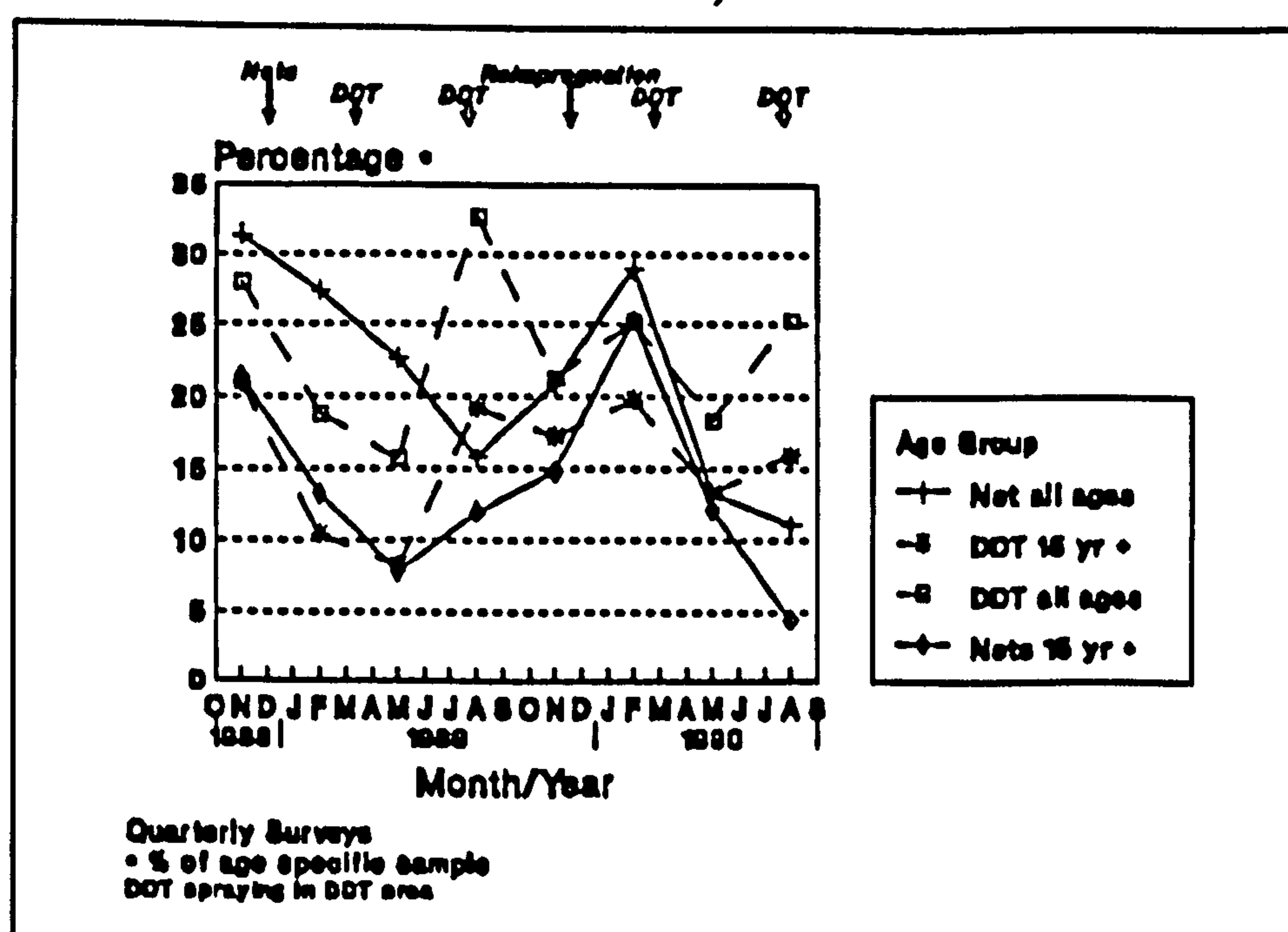
25% in the last survey. DDT spraying may have some impact on this age group. The rate in the



same age group in the bednet area did not decline immediately after bednets were introduced, till the fourth survey. It rose again thereafter but declined further after re-impregnation to about 10%. The rate in the 10-14 year old in the DDT area declined initially, but increased gradually to 10% more than the initial level. These increases in the DDT area were mainly with P.falciparum. The rate in the same age group in the bednet area, initially rose, but declined slowly. The rises in this age group in later part was mainly due to P.vivax. The delay of a positive effect of bednets in this age group was due to low usage. This was later improved with health education.

The prevalence in the 15 years and older, and all ages combined, are as in Fig.33. The rates in the 15 years and older, initially the same in both areas, declined with both interventions. There was then a slow rise in the bednet area, the nets were re-impregnated and the rate declined

Fig.33 *Malaria prevalence in adults and all ages combined in the bednet and DDT trial areas, Guadalcanal One.*



to 5%. The rate in the DDT area rose more sharply and was maintained between 15% and 20% throughout the study period. This high prevalence in the DDT area was mainly due to P.falciparum, while the few cases in the bednet area were P.vivax.



The rates in all ages combined in both DDT and bednet intervention areas, initially similar, declined with interventions. The decline in the DDT area was much faster but rose again till after the second DDT spraying cycle before dropping to maintain a prevalence between 20% and 25%. The rate in the bednet area rose eight months after nets were distributed but declined again after the nets were reimpregnated. The spike in early 1990 was due to P.vivax cases in the older children, mainly 10-14 year old. A.punctulatus was the main vector in this trial area. The general prevalence in the DDT area seemed to have declined slightly, but the ratio of P.falciparum to P.vivax changed from 1:1 at the beginning to 2.7 :1 at the end. The decline was not maintained and more cases still occurred in the younger age groups.

In the bednets group, the prevalence in general was initially similar to that in the DDT area. The majority of cases at the start was P.falciparum with 5.5 cases to one of P.vivax. But by the end there were more P.vivax, 1.6 to one of P.falciparum. The majority of cases in the end (Table 16) occurred in older age groups who were more likely to be infected outside bednets, or outdoors. It may be that with subsequent reduction of A.punctulatus older people wandered more outdoors and were infected by young adult vectors, with infective P.vivax, in particular A.farauti.

**Table 16.** Age specific malaria prevalence (%) by the last survey, Guadalcanal One trial area.

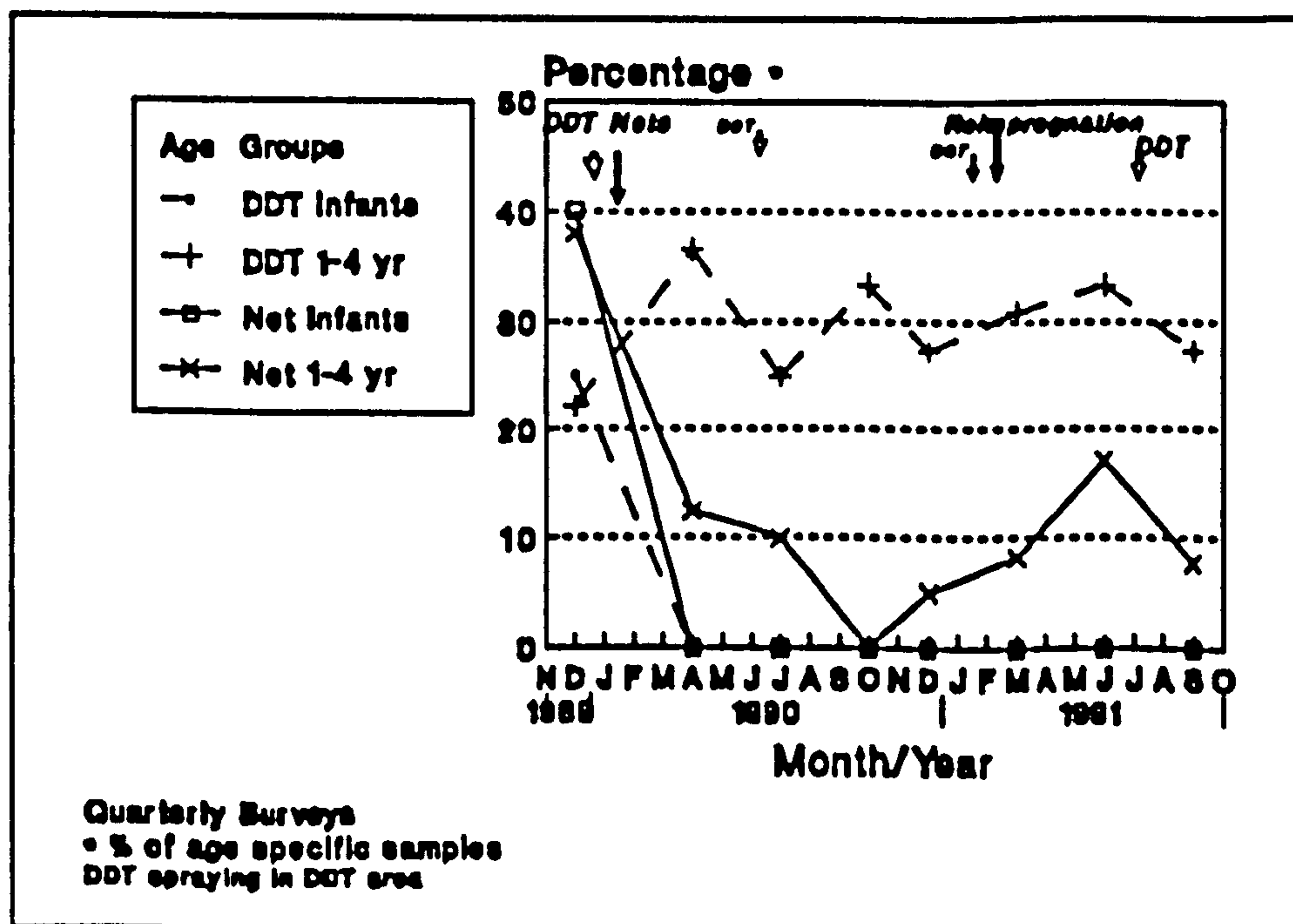
AGE GROUP	DDT				BEDNET			
	Number	Rate	P.fal	P.viv	Number	Rate	P.fal	P.viv
Infant	11	27.3	9.1	18.2	14	0	0	0
1-4	47	48.9	38.3	10.6	41	14.6	4.9	9.8
5-9	55	29.1	23.6	5.5	57	8.8	1.8	7.0
10-14	44	27.3	18.2	9.1	50	26.0	10.0	16.0
15 plus	126	15.9	11.1	4.8	91	4.4	3.3	1.1
TOTAL	293	25.3	18.4	6.8	253	11.1	4.3	6.7

The immediate positive effect with DDT was most likely due to an initial effective impact on A.punctulatus, but transmission, especially with P.falciparum, was re-stabilised again with A.farauti taking over as the main vector. The other possible reason was increased refusals to DDT spraying, thus after a decline in density, A.punctulatus may increase again maintaining transmission.

#### 4.2.7 Guadalcanal Island Two.

The prevalence in infants and 1-4 year old in the second trial area in Guadalcanal is presented in Fig.34. The infants rate immediately dropped to zero in both intervention areas and were maintained as such throughout the study. The rate in the 1-4 year old in the DDT area rose slightly and was maintained at 30% prevalence throughout the study with P.falciparum the main species. The rate in the same age group in the bednets area declined with impregnated bednets, to zero by the sixth month. It then rose slightly, mainly with P.vivax, but declined again, after the nets were reimpregnated, to less than 10%.

**Fig.34 Malaria prevalence in the under fives in bednet and DDT trial areas, Guadalcanal Two.**

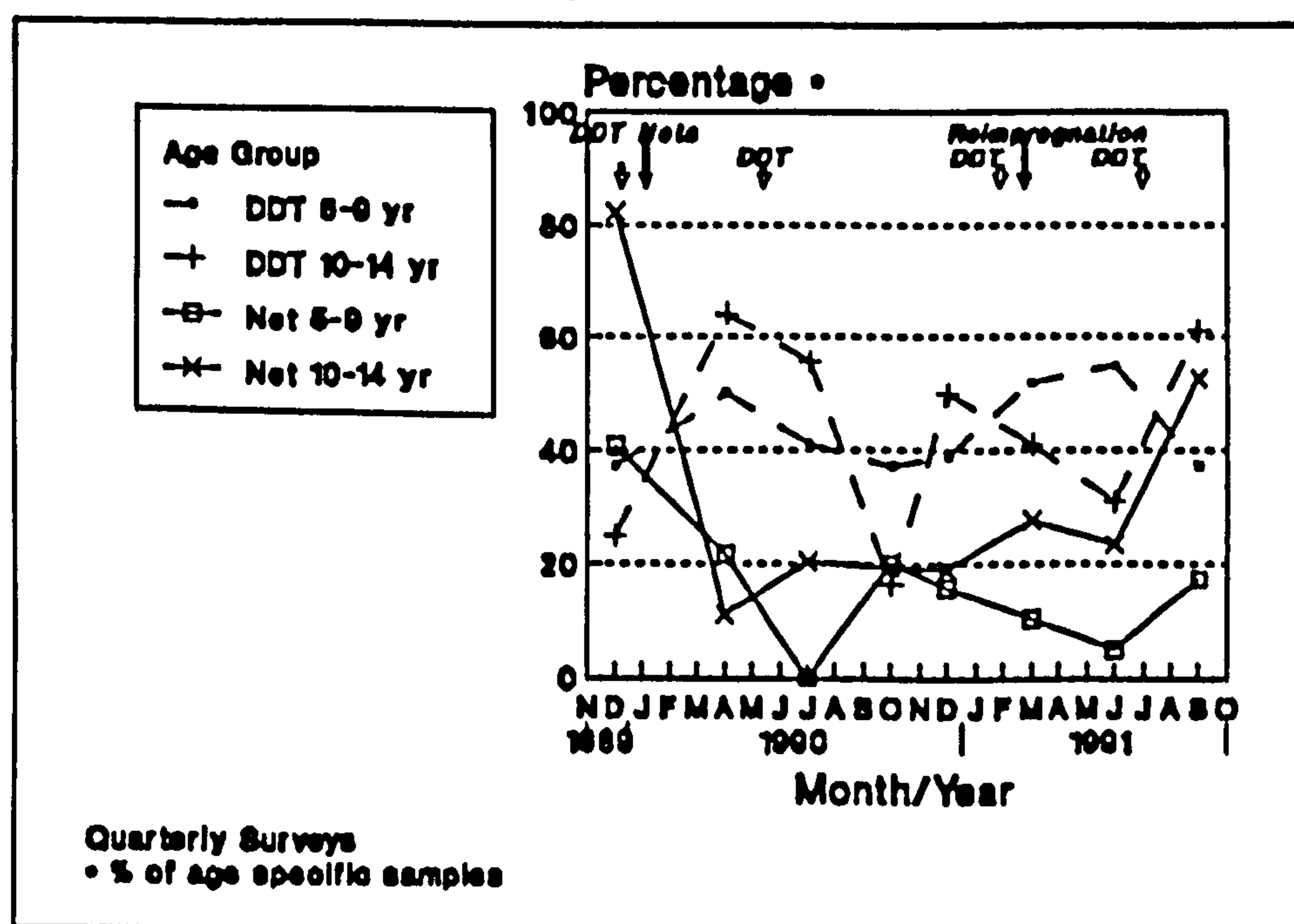


The prevalence in 5-9 year and 10-14 year old in this trial area is presented in Fig.35.



The rates in both age groups in the DDT area rose slightly and were maintained at level between 40% and 60%. There were variations which may be due to DDT spraying, but a positive impact was not sustained. The prevailing high prevalence was with both P.falciparum and P.vivax. The rate in the 5-9 year old in the bednet area, declined with the intervention and then rose slightly seven months after nets were distributed. It dropped again with re-impregnation of nets to less than the 20% level.

**Fig.35 Malaria prevalence in the 5 to 14 year old, in the bednet and DDT trial areas, Guadalcanal Two.**



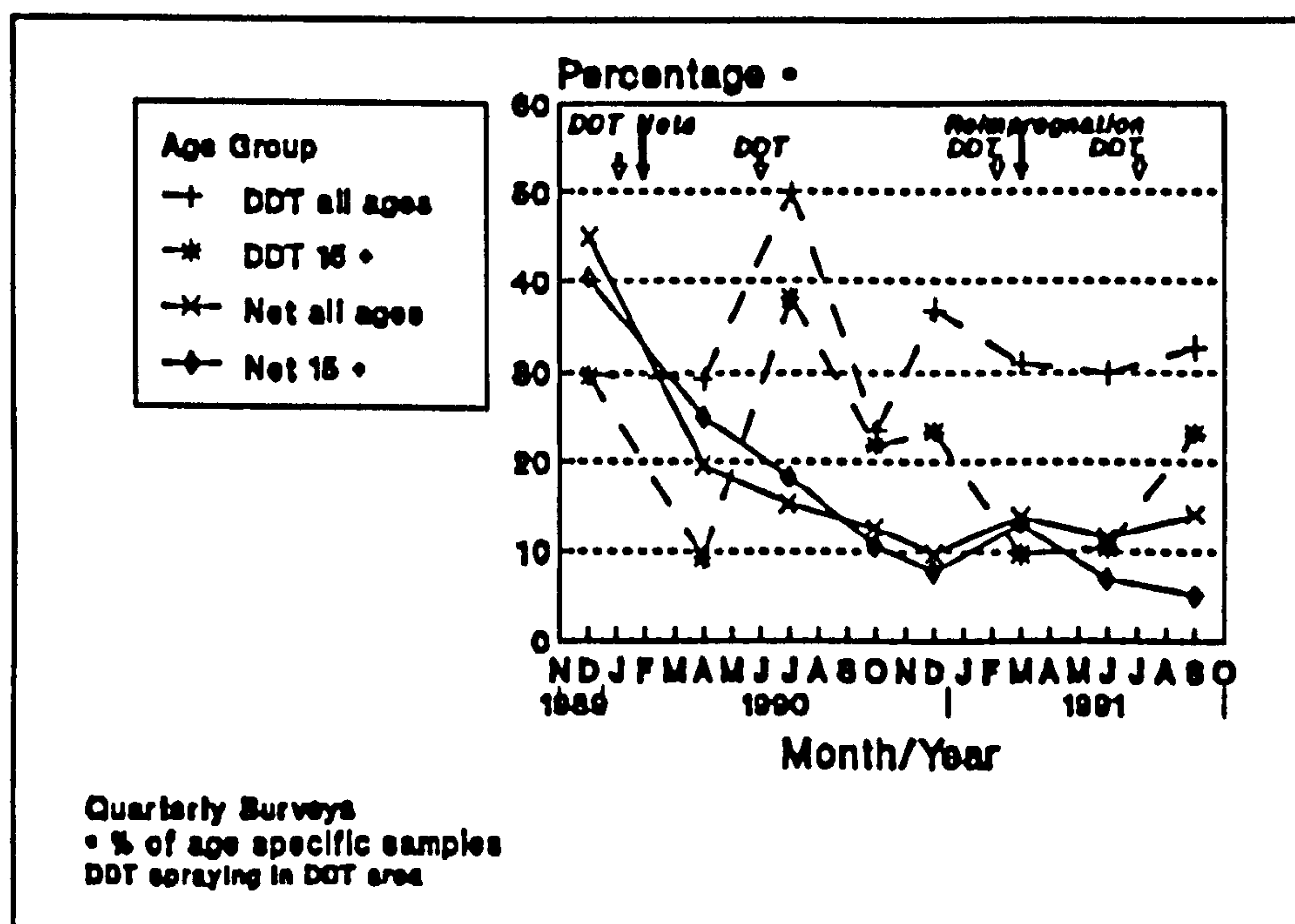
The rate in the 10-14 year old was initially very high at 80% in the bednets area. It dropped immediately with impregnated bednets and was maintained at around the 10% level till the last survey when there was an increase with a P.vivax outbreak. The prevalence in the later part of the study was with P.vivax.

The prevalence in the 15 years and older, and all ages combined are presented in Fig.36. The rate in the 15 years and older in the DDT area rose quickly after an initial drop, and fluctuated at the 20% level, mainly with P.falciparum infection. The rate in the bednet area with the same age group declined with impregnated nets. The slight rise prior to re-impregnation of



nets was curtailed with re-impregnation, and the rate declined to less than 10%. The rates in all ages combined in the DDT area, after a brief decline, rose quickly due to increases in the younger age groups. It dropped again and was maintained at a level of 30%. Transmission of P.falciparum in particular was maintained despite continued DDT spraying. The combined rate in the bednet area declined steadily and was maintained at 10% level with a spike in the last survey due to some cases of P.vivax in the 10-14 year old. It was mainly P.vivax that contributed to prevalence which was low in the bednet area.

**Fig.36 Malaria prevalence in adults and all ages combined in the bednet and DDT trial areas, Guadalcanal Two.**



The rates in the DDT area did not appear to change throughout the study. This was an area of high DDT refusals in the past. The parasite ratio at the start was 1.5 P.falciparum to 1 P.vivax, and the species remained equal at the end. In the bednets area, even though the rates were initially higher, there were reductions after impregnated bednets were introduced. The reduction was especially marked in the younger age groups particularly with P.falciparum. At the beginning for every 1.2 P.falciparum there was one P.vivax, but at the end this reversed to one P.falciparum to 2.2 P.vivax.

In the last survey (Table 17), most cases in the bednets area occurred in the older age groups, and especially with P.vivax. It was likely that they were inoculated outside bednets, especially outdoors by A.farauti with infective P.vivax sporozoites.

**Table 17.** Age specific malaria prevalence (%) by the last survey, Guadalcanal Two Trial area.

AGE GROUP	DDT				BEDNET			
	Number	Rate	P.falc.	P.viv.	Number	Rate	P.falc.	P.viv.
Infant	2	0	0	0	0	0	0	0
1-4	11	27.3	18.2	9.1	26	7.7	0	7.7
5-9	24	37.5	25	12.5	23	17.4	4.3	13.0
10-14	18	61.1	33.3	27.8	19	52.6	15.8	36.8
15 plus	52	23.1	5.8	17.3	59	5.1	3.4	1.7
TOTAL	107	32.7	15.9	16.8	132	14.4	4.5	9.9

#### 4.2.8 DDT Group versus Bednets Group.

When all data of both groups are analyzed together the results are as follows. The rates in all ages at the beginning of the study in both groups were as presented in Table 18. The *T* value derived from the sum of ranks between the two groups was not significant at 5% level. Therefore the null hypothesis was accepted as plausible and there was no difference between the two groups at the outset of the study, before impregnated bednets were introduced.

Eighteen to twenty four months years after the introduction of permethrin treated bednets the prevalence rates in all ages of the two groups are presented in Table 19. When the non-parametric test, *Wilcoxon Rank Sum Test*, is applied, the difference between the two intervention groups was statistically significant ( $p < 0.01$ ). Therefore permethrin treated bednets were more effective in reducing the prevalence of malaria than DDT at 1% significant level. When the prevalence of age specific groups are compared between the intervention areas, the differences are much more marked as follows. When all samples in infants were combined, as



**Table 18.** Prevalence rates in the two intervention groups prior to introduction of bednets.

PAIR	SITE	INTERVENTION GROUPS			
		DDT	RANK	BEDNET	RANK
1	Isabel	34.2	11	15.6	3
2	Vella	17.6	4	13.5	1
3	Vona/Kolo	14.3	2	29.1	7
4	Florida	29.6	8.5	19.3	5
5	Malaita	54.1	13	61.0	14
6	Guad One	28.0	6	31.4	10
7	Guad Two	29.6	8.5	44.9	12
SUM OF RANKS			53		52

**Table 19.** Prevalence rates in the two intervention groups two years after introduction of bednets.

PAIR	SITES	INTERVENTION GROUPS			RANK
		DDT	RANK	BEDNETS	
1	Isabel	32.7	11.5	10.7	3
2	Vella	25.0	8	8.3	1
3	Vona/Kolo	25.6	10	9.5	2
4	Florida	34.6	13	12.3	6
5	Malaita	46.9	14	11.8	5
6	Guadal1	25.3	9	11.1	4
7	Guadal2	32.7	11.5	14.4	7
SUM OF RANKS			77		28

in Table 20, the prevalence in the DDT area was higher than those in the bednets group especially with P.falciparum. The difference was significant ( $p < 0.001$ ). Permethrin impregnated bednets were very effective in protecting infants against malaria by an estimated fraction of



77.86%, especially P.falciparum (87.95%).

In the 1 to 4 year old the total samples in the intervention areas are presented in Table 21. The rates in the bednet group were much lower than in the DDT area. These differences, were statistically significant ( $p < 0.001$ ). Permethrin impregnated bednet was much more effective in reducing malaria prevalence in this age group than DDT house spraying, especially against P.falciparum.

**Table 20.** Total sample, positive and species distribution, in infants, all surveys in the intervention groups.

INTERVENTIONS	Total sample	Positive (%)	<u>P.falciparum</u> (%)	<u>P.vivax</u> (%)
DDT	534	21.91	14.61	7.30
Impregnated bednets	454	4.85	1.76	3.09

**Table 21.** Total sample in the 1-4 yr old, positive and species distribution, in all surveys in the intervention groups.

INTERVENTIONS	Total sample	Positive (%)	<u>P.falciparum</u> (%)	<u>P.vivax</u> (%)
DDT	2597	33.50	22.56	10.94
Impregnated bednets	1917	15.96	6.26	9.70

The total samples and results in the 5 to 9 year old in the study are summarised in Table 22. The positive rate in the bednet group was lower than in the DDT area by half. This difference which is also marked with P.falciparum was again quite significant ( $p < 0.001$ ). Permethrin treated bednets are therefore much more effective in keeping a low malaria prevalence in this age group than DDT residual house spraying.

The total samples in the 10 to 14 year old are summarised in Table 23. The positive rate

in the bednet group was lower than in the DDT group. Even though the relative rate in this group is less than in other age groups, the difference is still statistically significant ( $p < 0.001$ ). This was still marked with P.falciparum. However in this age group there was no advantage in

**Table 22.** Total sample in 5-9 yr old, positive and species distribution in all surveys in the intervention groups.

INTERVENTIONS	Total sample	Positive (%)	<u>P.falciparum</u> (%)	<u>P.vivax</u> (%)
DDT	3166	31.11	19.17	11.94
Impregnated bednets	2508	15.43	6.86	8.57

protection against P.vivax by permethrin impregnated bednet. The older children were therefore much more likely to be still inoculated outside the bednet, especially outdoors.

The total samples in 15 years and older and the results are summarised in Table 24. Permethrin impregnated bednets maintained a lower prevalence than DDT spraying. There is

**Table 23.** Total sample in 10-14 yr old, positive and species distribution in all surveys in the intervention groups.

INTERVENTIONS	Total sample	Positive (%)	<u>P.falciparum</u> (%)	<u>P.vivax</u> (%)
DDT	3062	24.59	14.59	10.38
Impregnated bednets	2445	18.86	7.53	11.33

again a statistical significance in the difference ( $p < 0.005$ ). The main difference between the intervention was due to P.falciparum, even though in this adult age group bednet appeared to provide a similar protection to both species, and almost similar protection against P.vivax as DDT spraying. The estimated fraction of malaria prevalence prevented by permethrin impregnated bednets are presented in Table 25. Permethrin impregnated bednets prevented



prevalence of infection in all ages, but the degree was much more with P.falciparum. It was

**Table 24.** Total sample in 15 yr and older, positive and species distribution, in all surveys in the intervention groups.

INTERVENTIONS	Total sample	Positive (%)	<u>P.falciparum</u> (%)	<u>P.vivax</u> (%)
DDT	7744	19.72	12.28	7.44
Impregnated bednets	6217	12.92	6.00	6.92

higher in the age groups under 10 years where more than half of the infections, as would have been with DDT residual spraying, were prevented. In those below 10 years over 60% of P.falciparum infection were prevented by impregnated bednets. In the older age groups the degree of protection was lower, and even though permethrin impregnated bednets was still very effective against P.falciparum, there were no marked benefits against P.vivax.

**Table 25.** Estimated prevented fraction (%) of malaria prevalence by permethrin impregnated bednets over DDT spraying in age specific groups.

AGE GROUPS	PREVENTED FRACTION (%)	
	All species	<u>P.falciparum</u>
Infants	77.86	87.95
1 - 4 yr	52.36	72.26
5 - 9 yr	50.40	64.21
10 - 14 yr	23.30	48.39
15 yr plus	34.48	51.14

### 4.3 CHANGES IN INCIDENCE OF MALARIA INFECTIONS.

#### 4.3.1 Introduction

The results on incidence of infection in the study areas are presented in the following



sections. Compliance to DDT spraying in these areas were high in the past and so were the initial low incidence parts of the trial areas. This was monitored by the efficient PCD blood slide mechanism which has been established and is in operation.

#### 4.3.2 Isabel Island

The incidence in infants and 1-4 year age groups in the bednet area is presented in Fig.37 and Fig.38 respectively. After the introduction of impregnated bednets it rapidly decreased to zero in the infants, except for a few sporadic cases, mainly of P.vivax. There were a few cases of P.falciparum which declined after re-impregnation. In the 1-4 year old the decrease

Fig 37: *Incidence of malaria in infants, bednet area, Isabel island.*

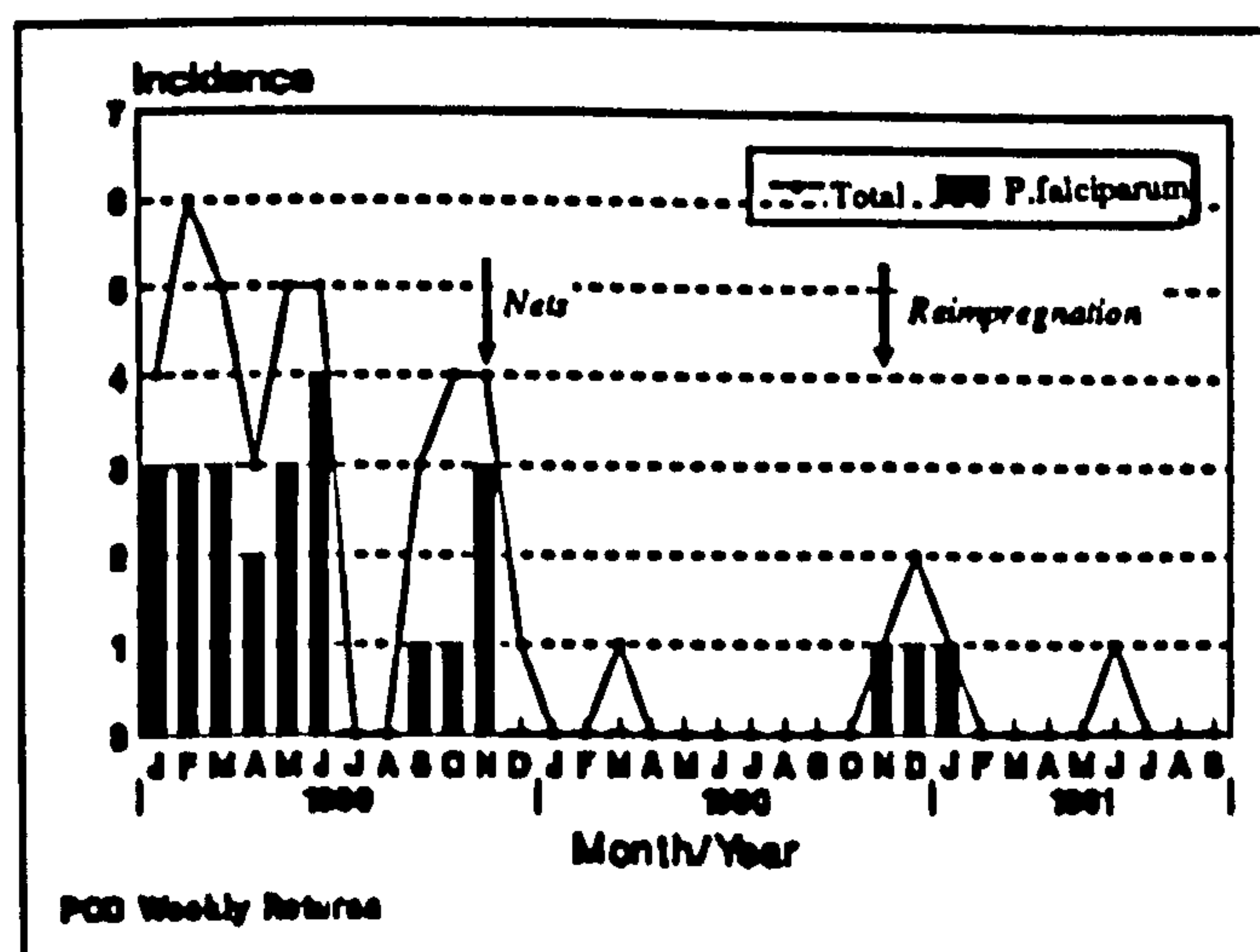
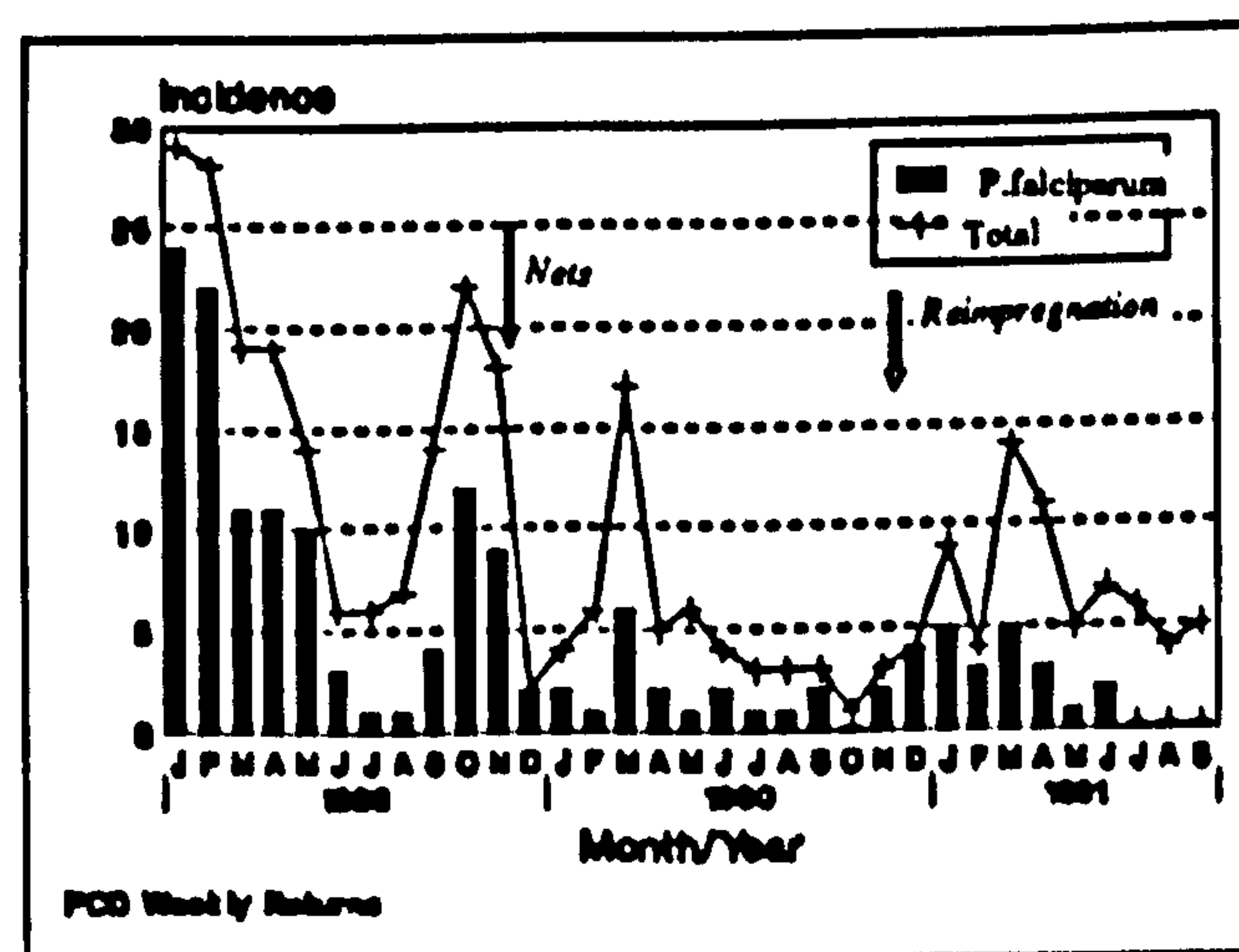


Fig.38: *Incidence of malaria in 1-4 yr old, bednet area, Isabel island.*



was immediate, especially with P.falciparum. The slight rise in P.falciparum in the first quarter after re-impregnation was not as high as prior to distribution of bednets. P.vivax was the main species after intervention. The reduction in mean monthly incidence in these two age groups were marked, especially with P.falciparum.

The results of the 5-9 year and 10-14 year olds are presented in Fig.39 and Fig.40. In the 5-9 year age group, the incidence dropped with impregnated bednets especially with

P.falciparum. The rise in the next peak transmission season was much lower than prior to the use of impregnated bednets, in particular with P.falciparum. The reduction in monthly incidence was marked in this group especially with P.falciparum, which was kept low. In the 10-14 year olds the reduction with impregnated bednets was dramatic, again mainly with P.falciparum. The

Fig.39: *Incidence of malaria in 5-9 yr old, bednets area, Isabel island*

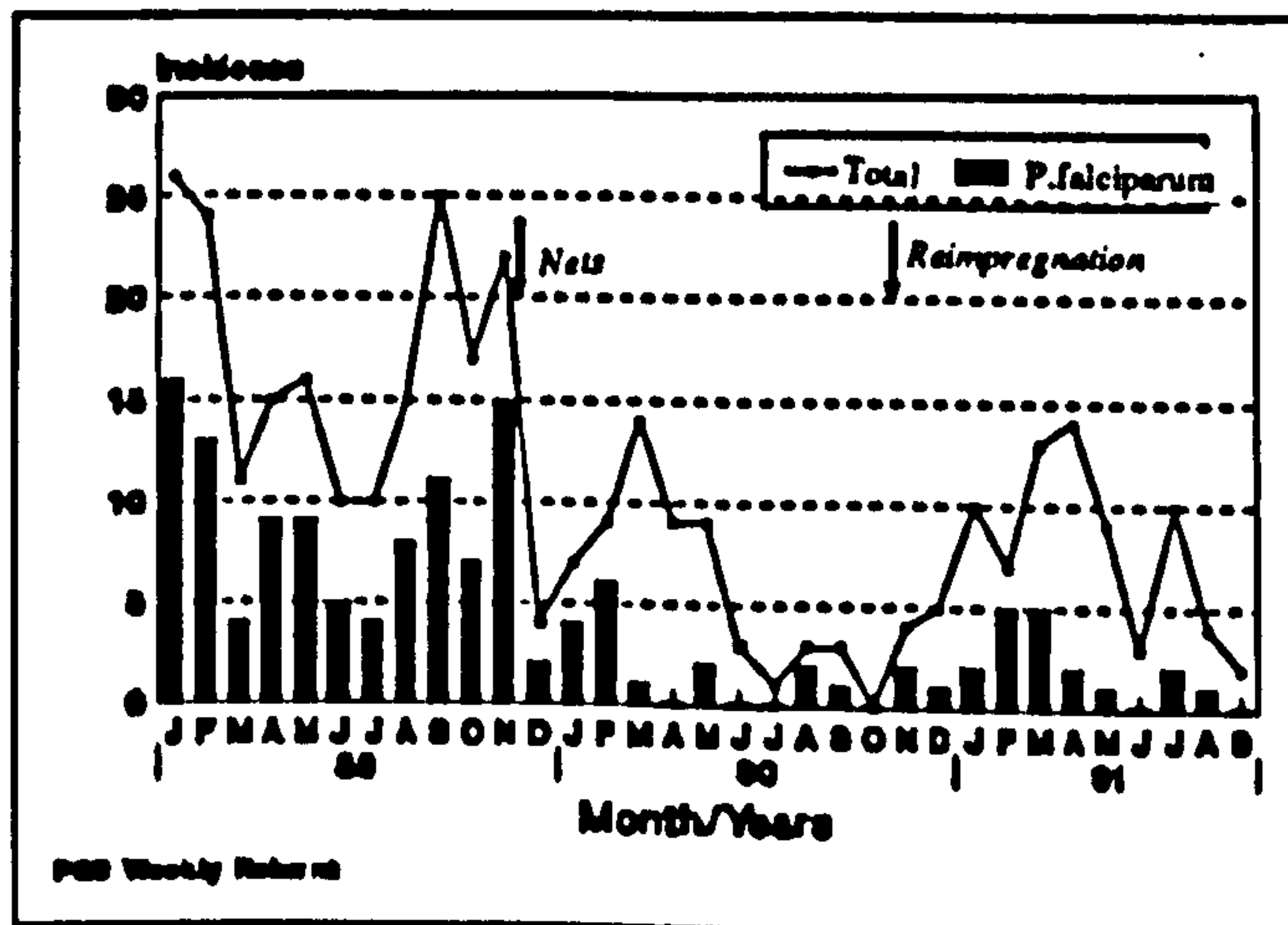
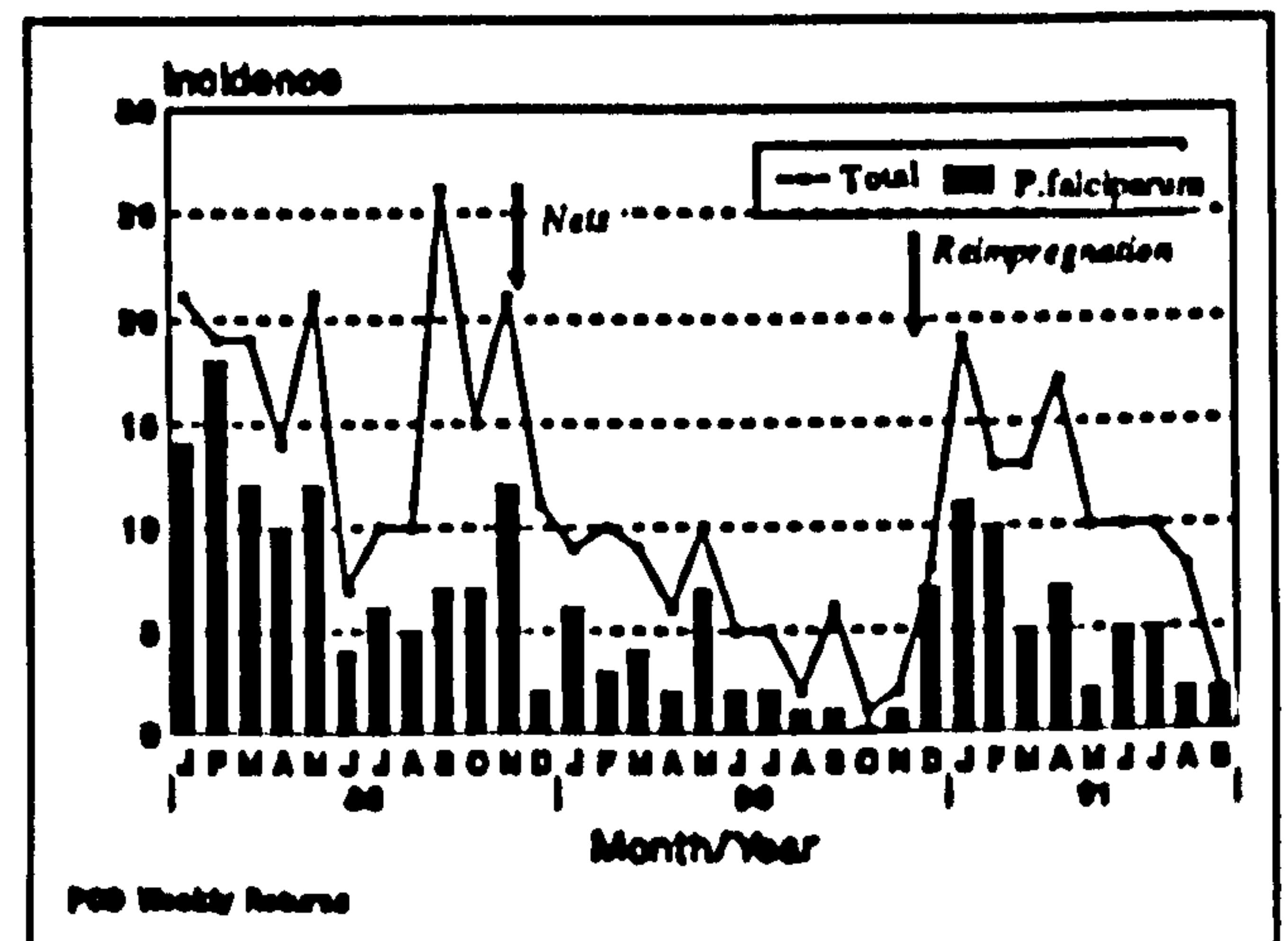


Fig.40: *Incidence of malaria in 10-14 yr old, bednet area, Isabel island*



rise in the first quarter of 1991 during the peak transmission season, even though lower than prior to bednet use, was higher than in the first quarter of 1990. This rise included P.falciparum. It was likely that older children stayed outside bednets, and even outdoors, allowing themselves to be inoculated, thus the reductions were not maintained as effectively as in the under 10 year age groups.

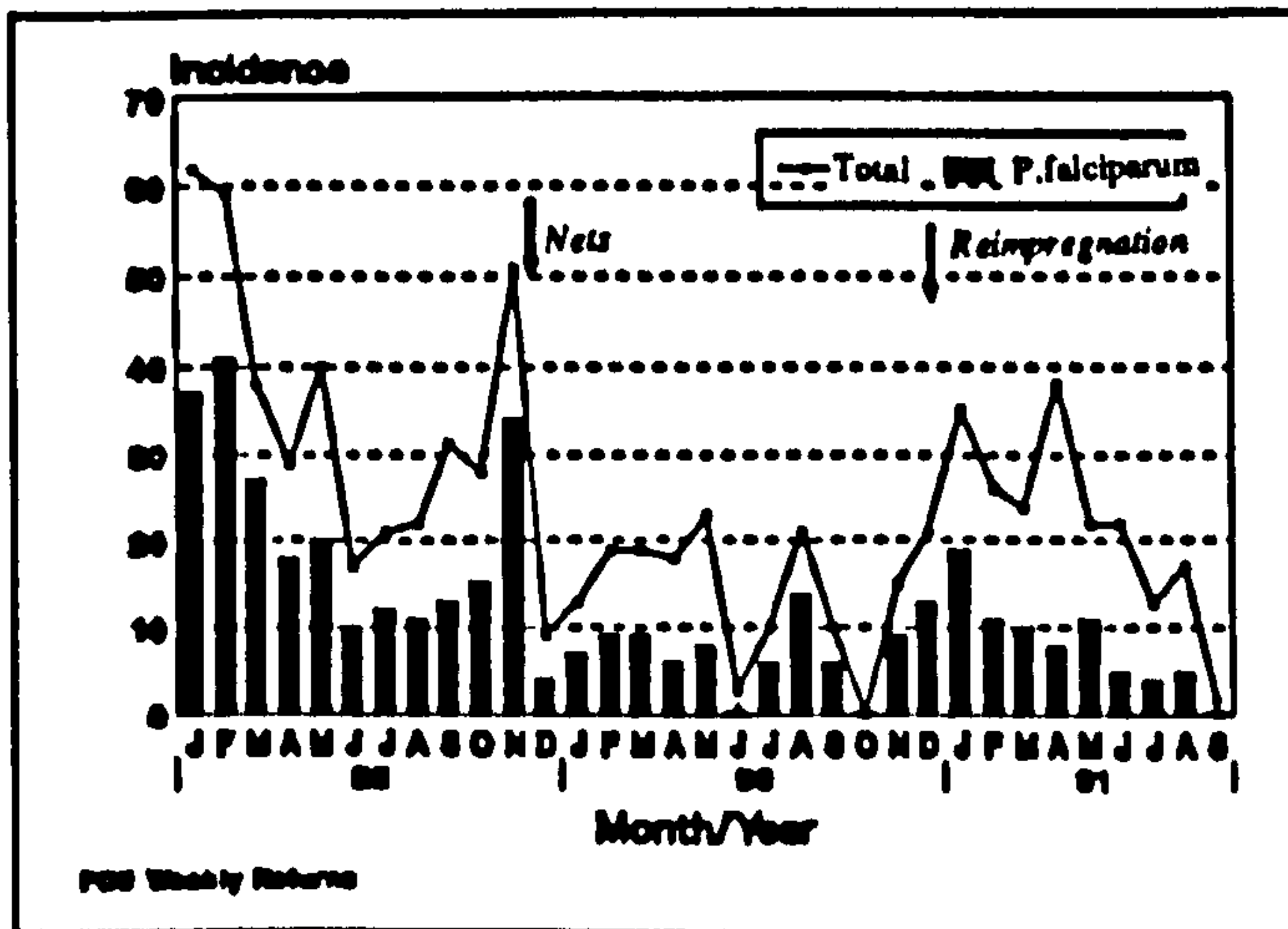
The results in the 15 years and older and in all ages combined, are as shown in Fig.41 and Fig.42. The incidence in the 15 years and older declined, especially with P.falciparum. The low level was maintained and the rise during the first quarter of 1991 was mainly due to P.vivax. These increases were not to the same level as prior to the introduction of bednets.

In all ages, there was an immediate decline with impregnated bednets, especially with P.falciparum. The rise during the peak transmission season in 1990 was curtailed, but that in the first quarter of 1991, even after re-impregnation, was mainly in the older age groups of 10 years

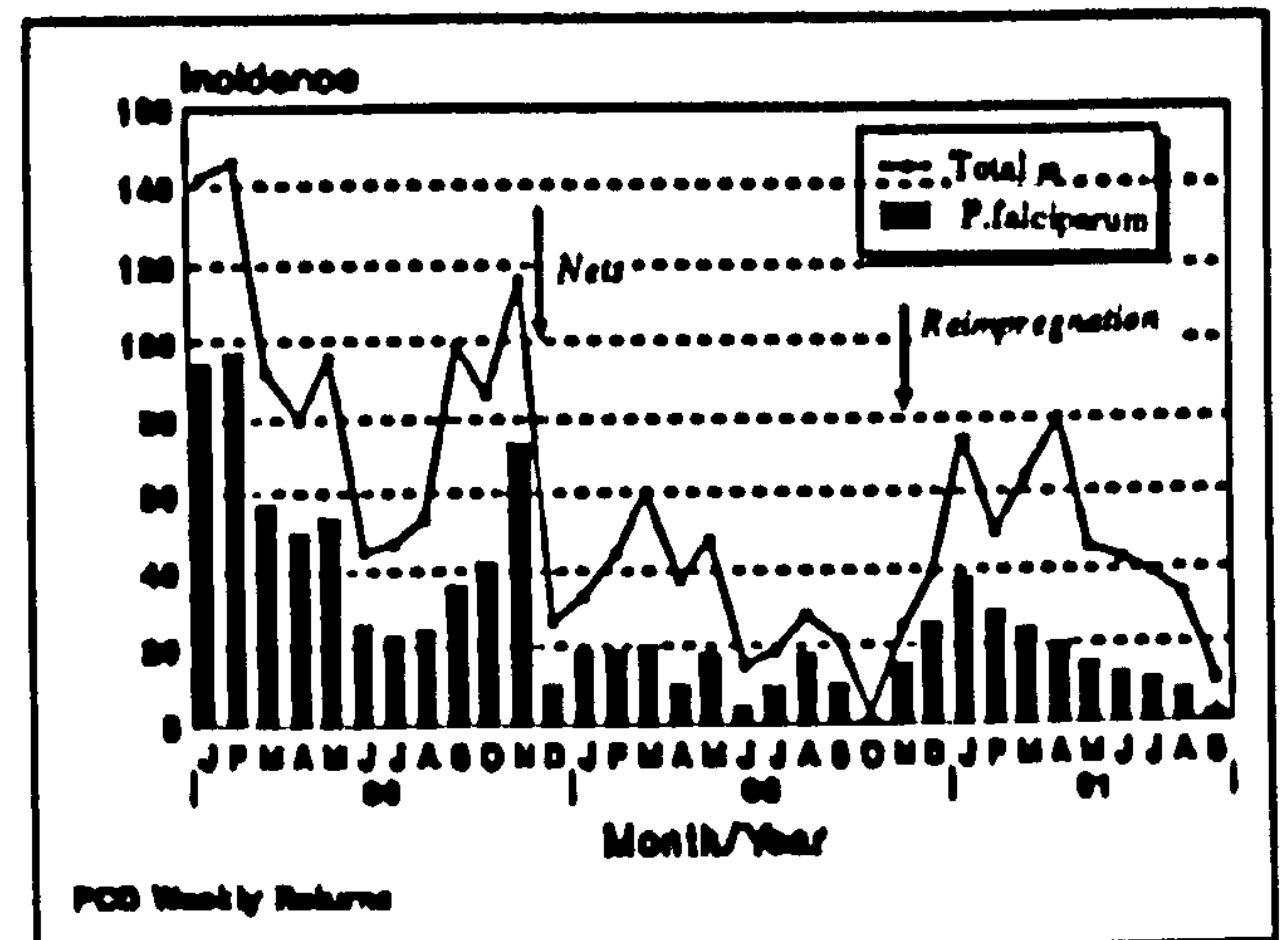


and older. The older age groups are likely to be infected outside bednets, especially when

**Fig.41:** *Incidence of malaria in 15 yr +, bednet area, Isabel island*



**Fig.42:** *Incidence of malaria in all ages, bed net area, Isabel island.*

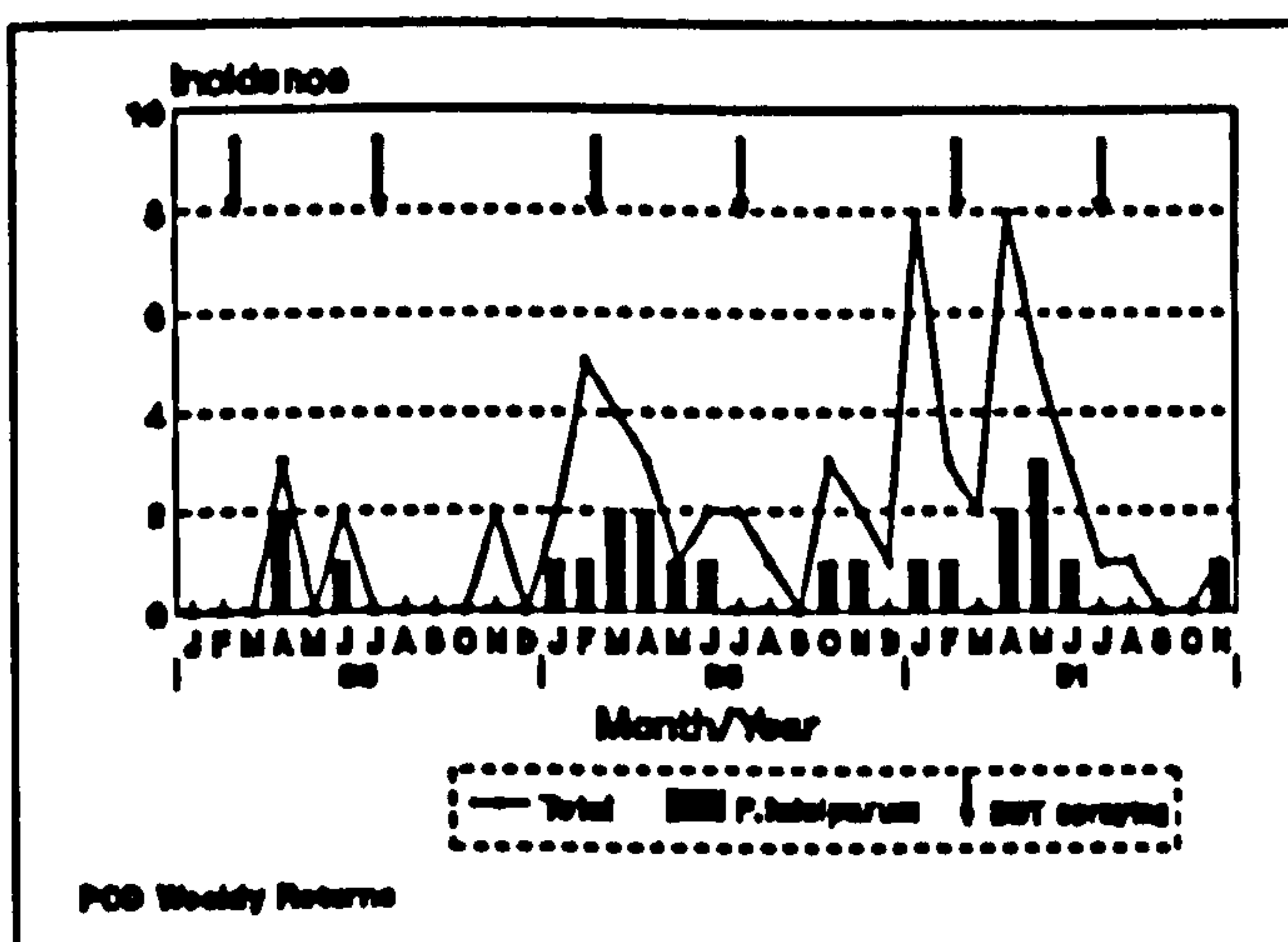


they are outdoors. The increase was mainly with

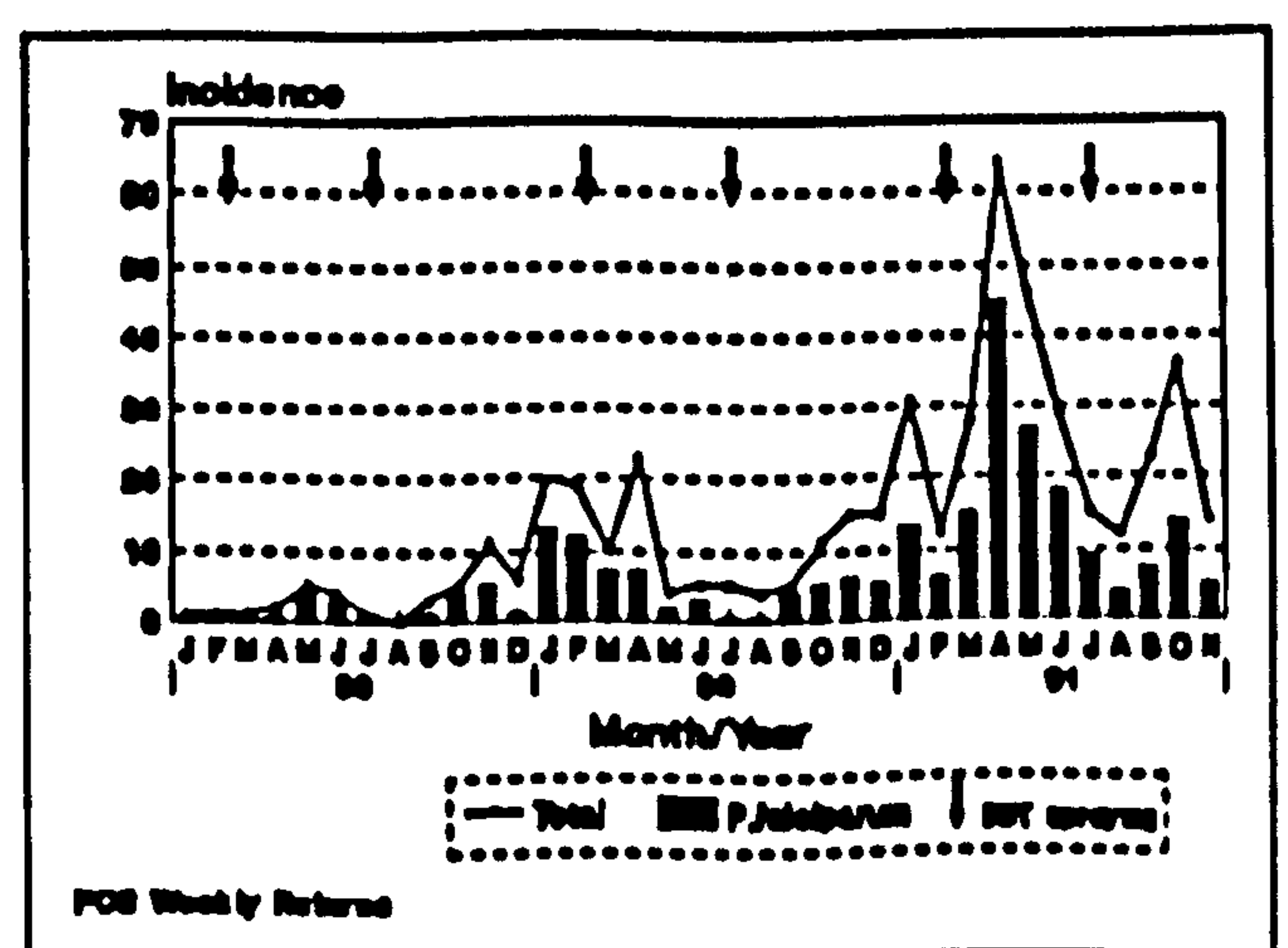
P.vivax, but not to the same level as prior to bednet introduction. The net effect on incidence is quite positive and especially in the under 10 year olds, and with P.falciparum.

The incidence in infants and 1-4 year old in the DDT area in Isabel island are respectively presented in Fig.43 and Fig.44 The incidence in infants was low prior to 1990, but since then there were increases especially with P.vivax. There were some cases of P.falciparum as well. The increase in overall incidence in infants is quite obvious, even though there were

**Fig.43:** *Incidence of malaria in infants, DDT area, Isabel island.*



**Fig.44:** *Incidence of malaria in 1-4 yr old, DDT area, Isabel island.*





only a few cases each month. The changes in incidence in the 1-4 year olds was also more marked. Even though the incidence in the eighties was maintained at a low level, it started to rise in 1990, markedly in 1991, especially with P.falciparum, during the peak transmission season. As with infants, any possible positive impact with continued DDT spraying was not sustained.

The changes with incidence in the 5-9 year and 10-14 year old are presented in Fig.45 and Fig.46. In the 5-9 year old, the incidence increased steadily, with peaks during the high transmission seasons in 1990 and 1991, despite continued DDT spraying. This increase was also with P.falciparum.

The increase in incidence in 10-14 year olds followed the same pattern especially with P.falciparum, and in epidemic proportions. There were very high incidences during the peak transmission season in the first quarter of each year from 1990 in this age group, the next peak significantly higher than the previous one, especially with P.falciparum malaria.

Fig.45: *Incidence of malaria in 5-9 yr old, DDT area, Isabel island.*

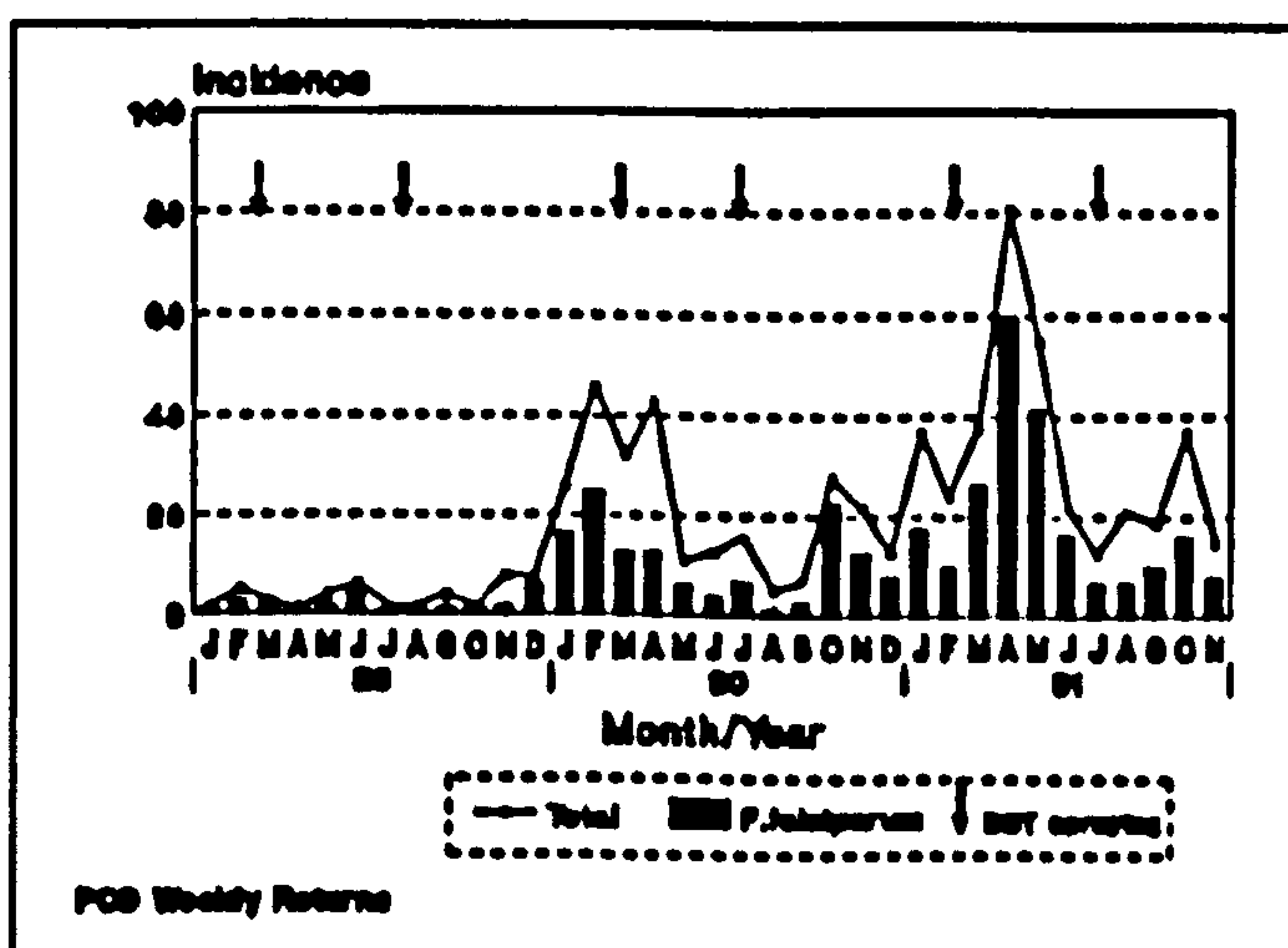
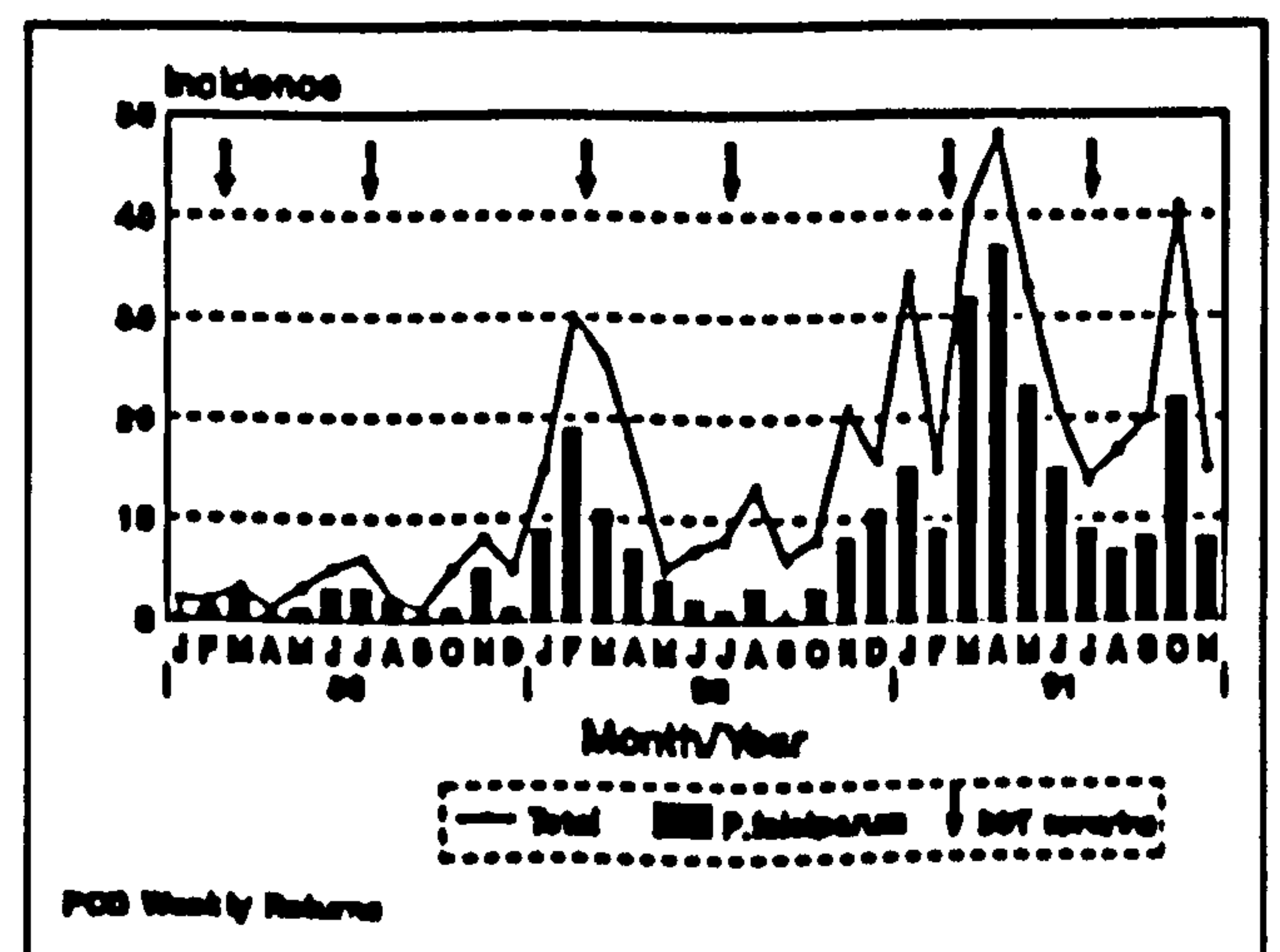


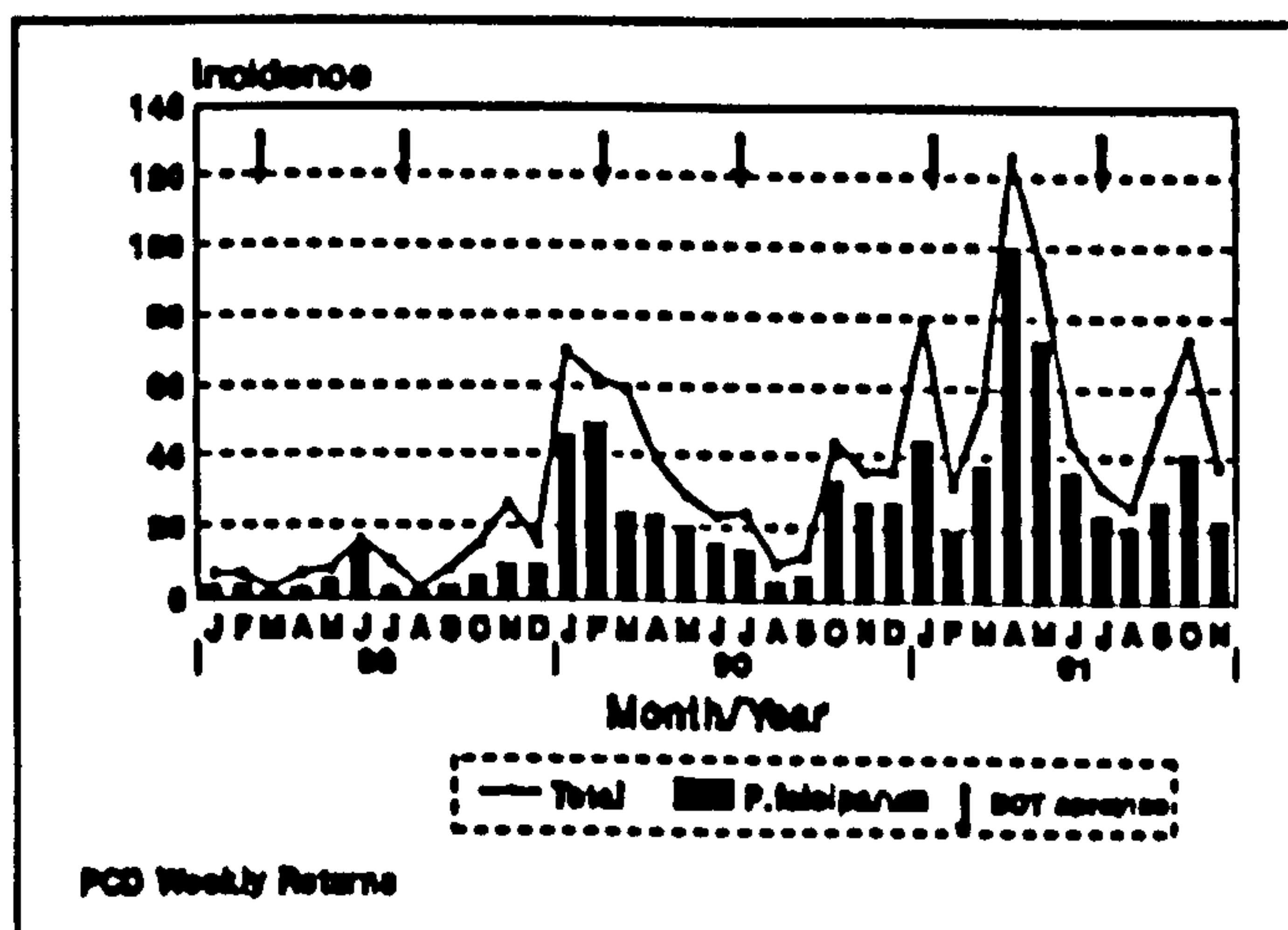
Fig.46: *Incidence of malaria in 10-14 yr old, DDT area, Isabel island.*



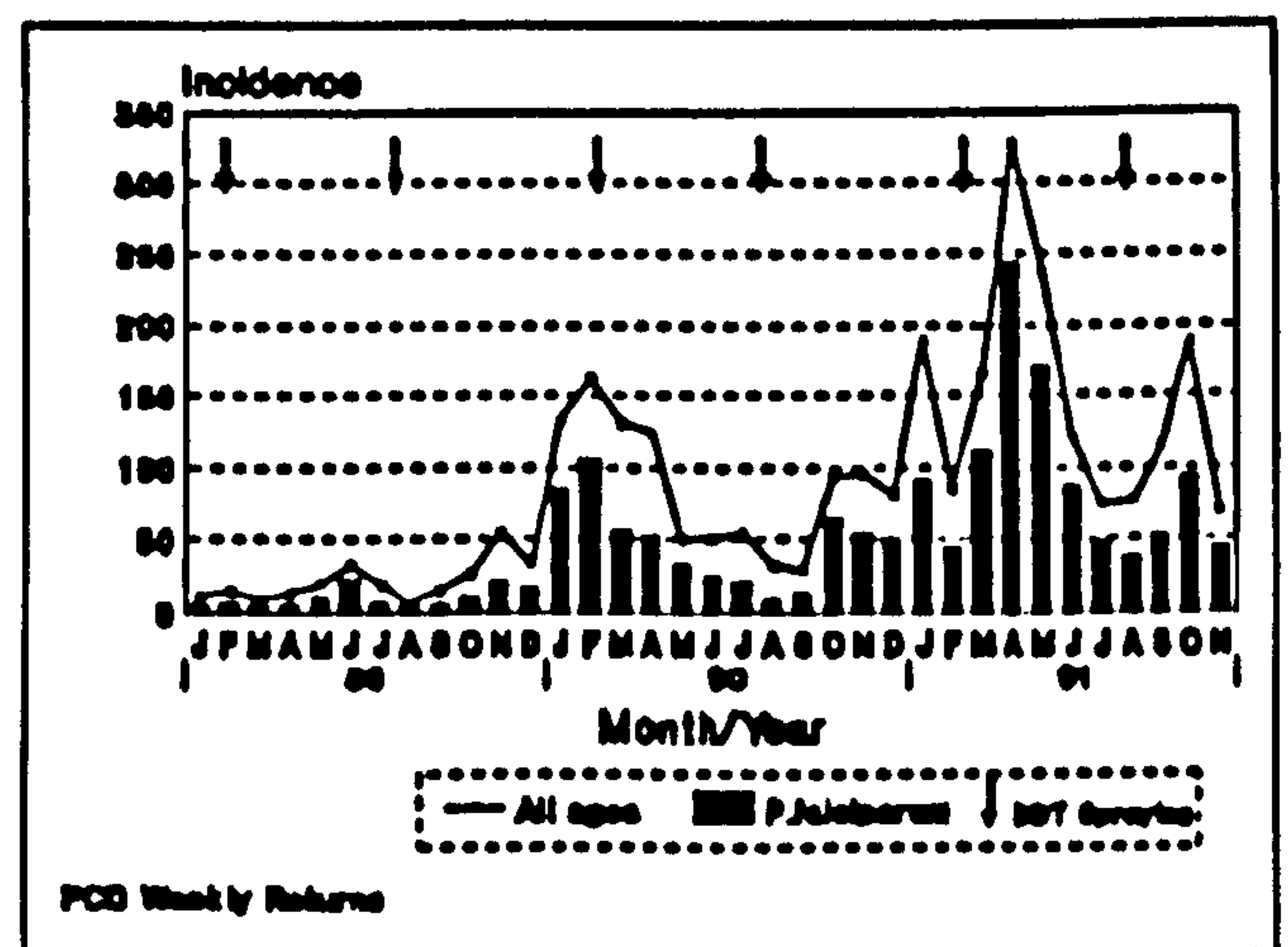
The changes with incidence in the 15 years and older, and in all ages combined in the DDT area are presented in Fig.47 and Fig.48. The pattern in the 15 years and older was similar to that of the 10-14 year old except with a higher number of cases. The increase was in epidemic

proportions, especially with P.falciparum. At this stage there was no apparent effect of DDT spraying. In all ages, even though the incidence was initially low, it increased steadily since 1990 with peaks during high transmission season in the first quarter of the year. These increases were marked in all ages, except infants, and especially with P.falciparum. It may be that infants, and the 1-4 year age group, to a certain extent, go to bed earlier inside sprayed rooms, or some may use bednets, even unimpregnated ones. The older age groups would be

**Fig.47:** *Incidence in 15 yrs.+, DDT area, Isabel island.*



**Fig.48:** *Incidence in all ages, DDT area, Isabel island.*



exposed to inoculations especially outdoors. After a period of reasonable control up to the eighties, even the adults were also susceptible to malaria infection. In this trial area it is the older age groups who were responsible for the increases in incidence when DDT spraying was apparently no longer effective.

### 4.3.3 Vona Vona/Kolobangara Islands

The changes in incidence of infection in this study trial area represented another different epidemiological situation. After a period of reasonable control in the seventies and eighties, epidemics occurred in the late eighties, mainly of P.falciparum. As a consequence ULV spraying was carried out around the island of Kolobangara in April 1989 as a remedial measure prior to the introduction of bednets in November 1989.



The changes in incidence in infants and 1-4 yr olds in the bednet area (Kolobangara) are presented in Fig.49 and Fig.50. In infants the incidence declined with ULV spraying but a further decrease was achieved with impregnated bednets. This reduction of the monthly incidence was marked, especially with P.falciparum. Few cases that were detected after intervention were of P.vivax and only two cases of P.falciparum in April 1991. In the 1-4 year olds a further important decrease was achieved with impregnated bednets, which maintained the monthly incidence of malaria at a low level throughout the study period.

Fig.49: *Incidence of malaria in infants, Kolobangara*

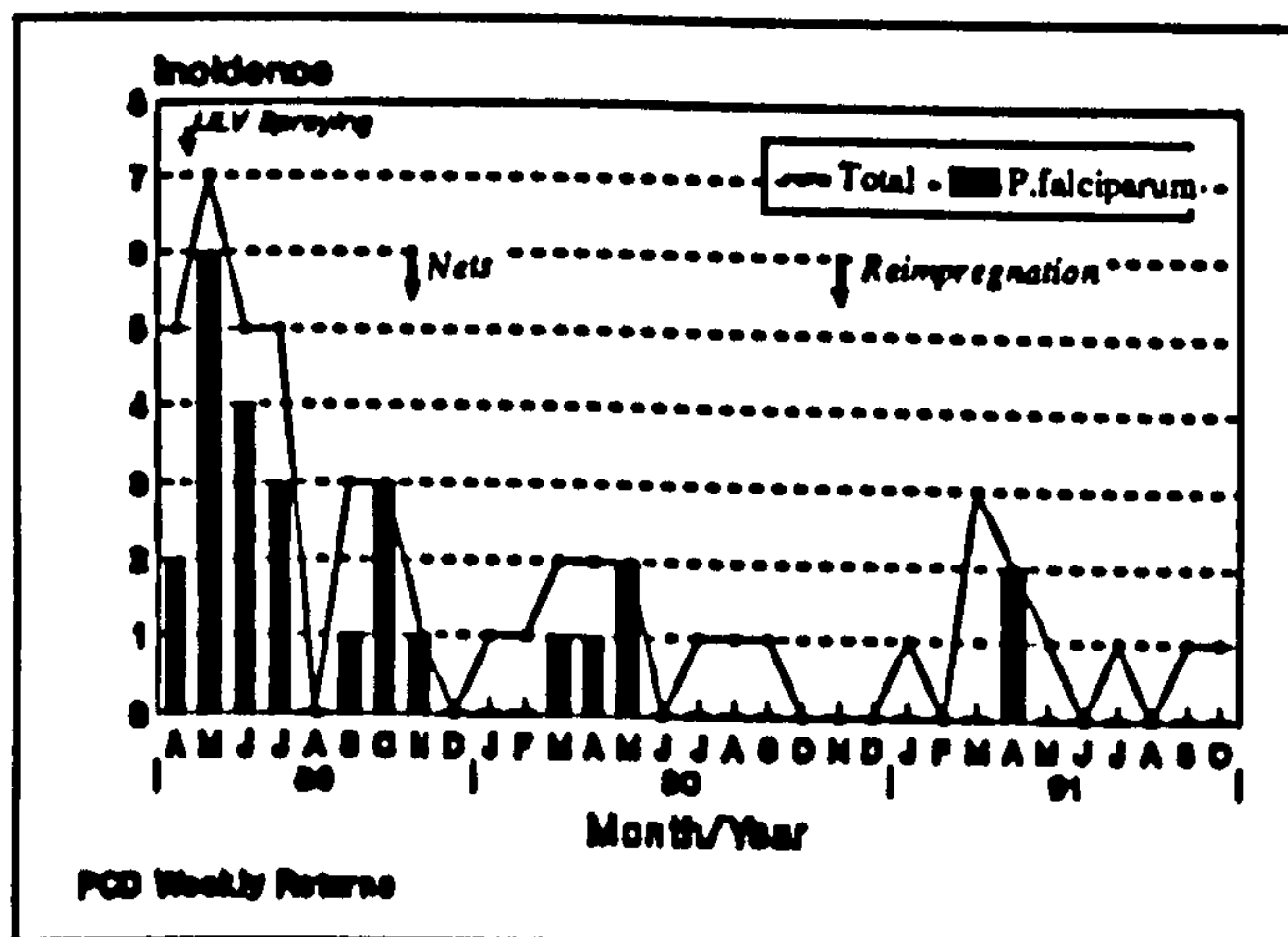
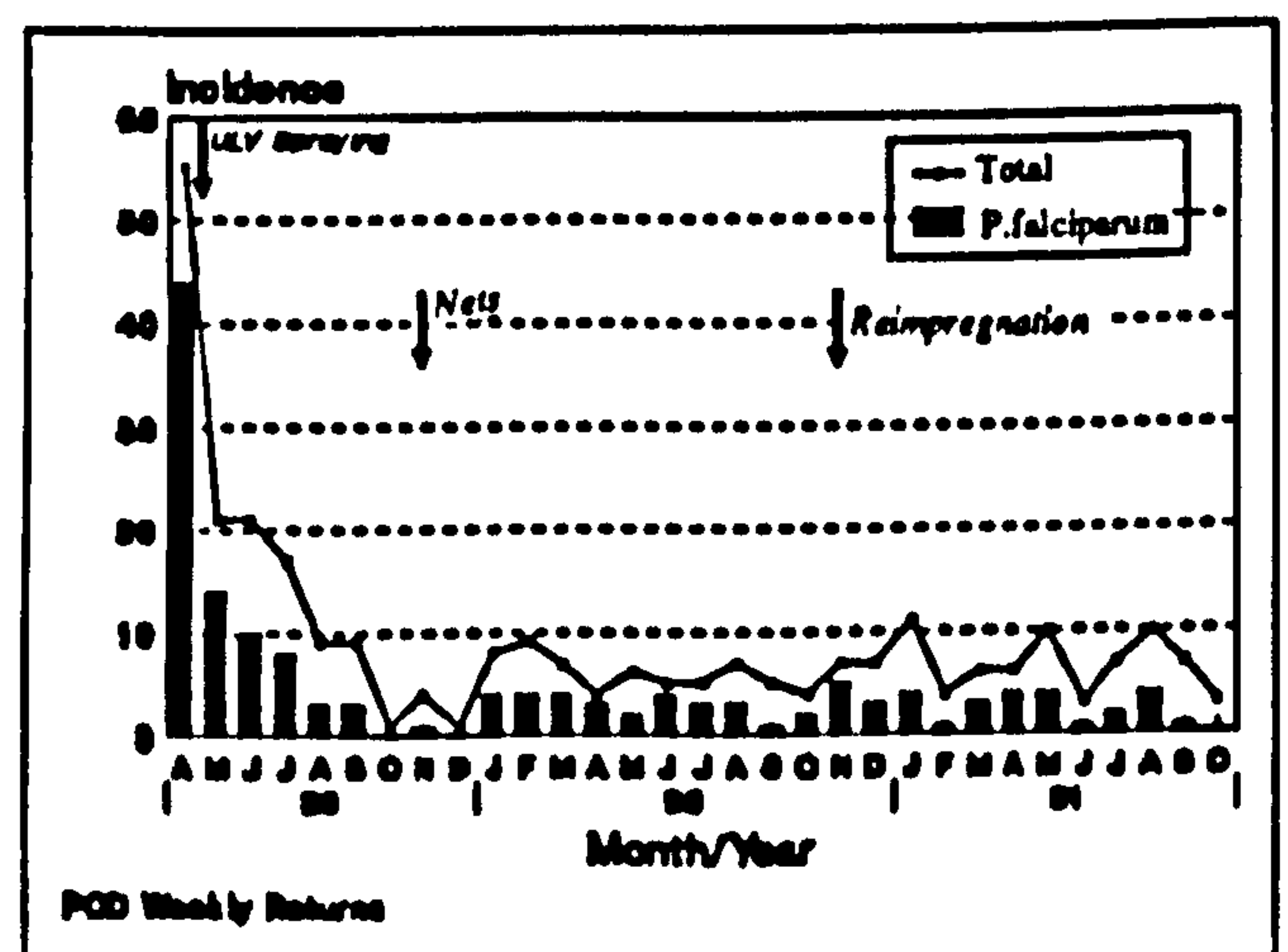


Fig.50: *Incidence of malaria in 1-4 yr old, Kolobangara*



The results in the 5-9 year and 10-14 year old are presented in Fig.51 and Fig.52. In the 5-9 year old further reductions were achieved with impregnated nets especially of P.falciparum. The low level was maintained with re-impregnation of nets and most cases that occurred were of P.vivax. The changes in monthly incidence in this age group prior to and after bednets, was quite marked, especially with P.falciparum. In the 10-14 year olds, a further significant reduction was also achieved with impregnated bednets. This low level was maintained throughout and into 1991.

The incidence in the 15 years and older, and all ages combined are presented in Fig.53 and Fig.54. In the 15 year olds a further reduction in incidence was achieved with impregnated bednets, especially with P.falciparum. The low incidence, even much more than in the younger



Fig.51: Incidence of malaria in 5-9 yr olds, Kolobangara

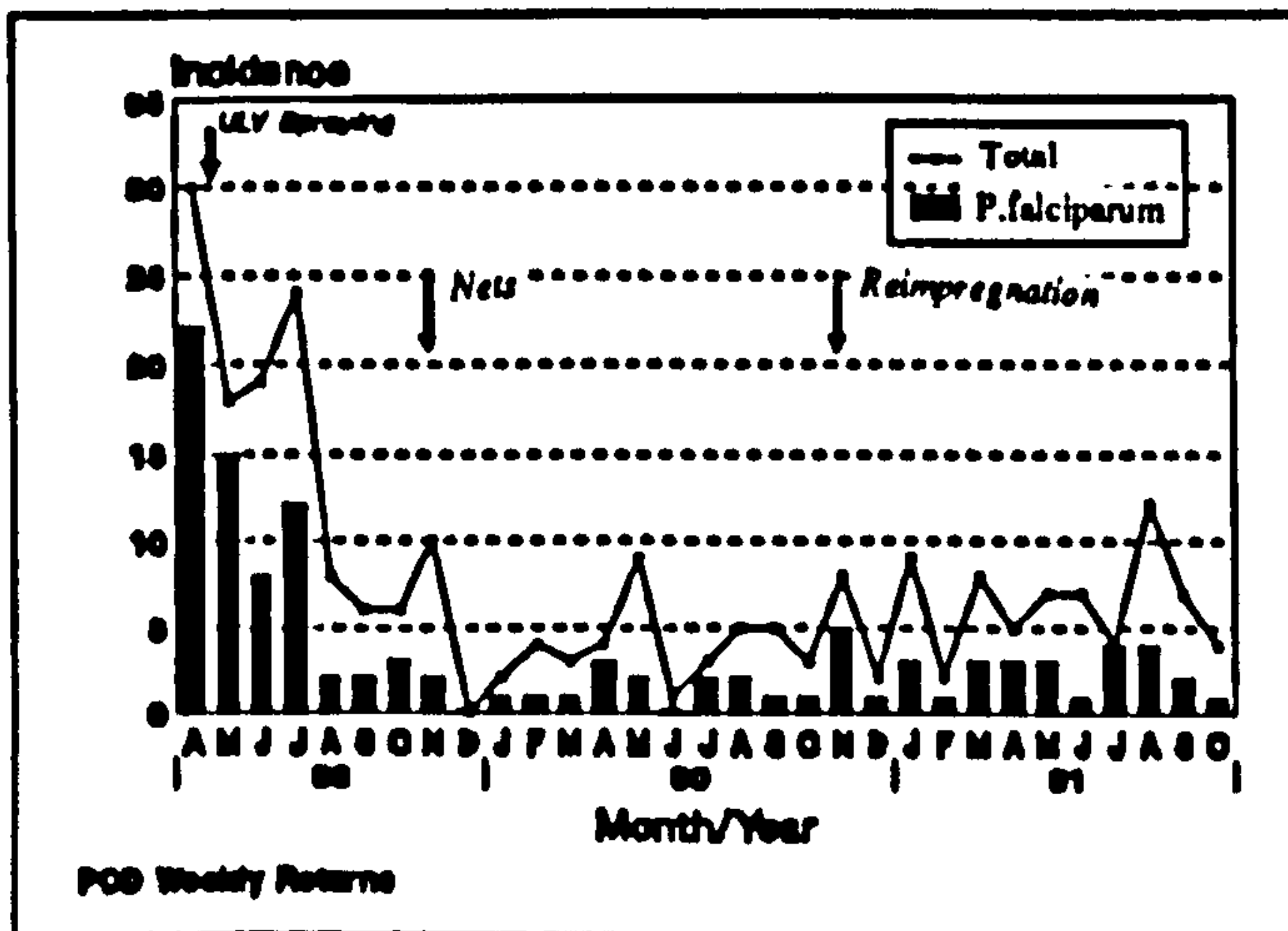


Fig.52: Incidence of malaria in 10-14 yr olds, Kolobangara

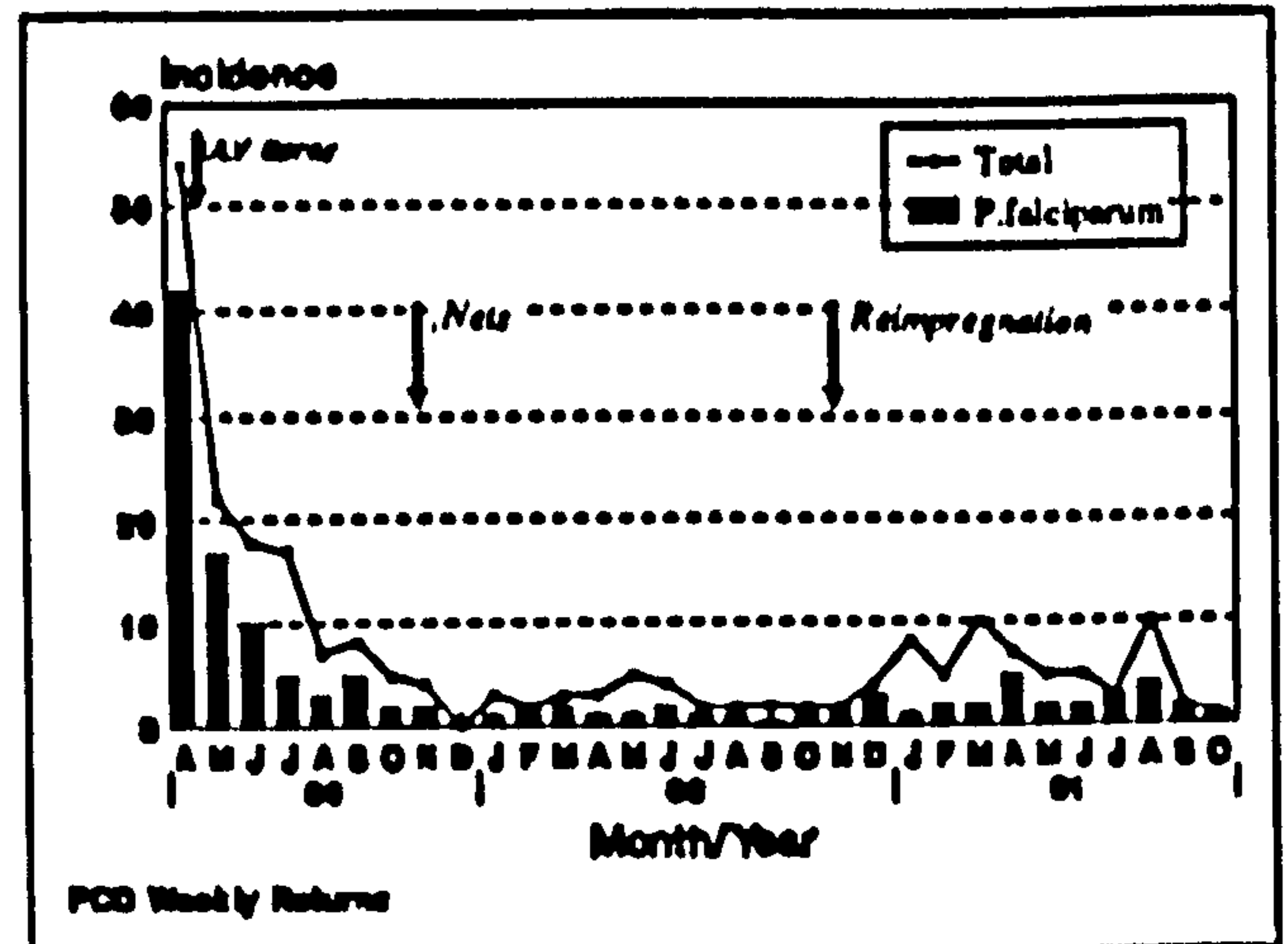


Fig.53: Incidence of malaria in 15 yrs +, Kolobangara.

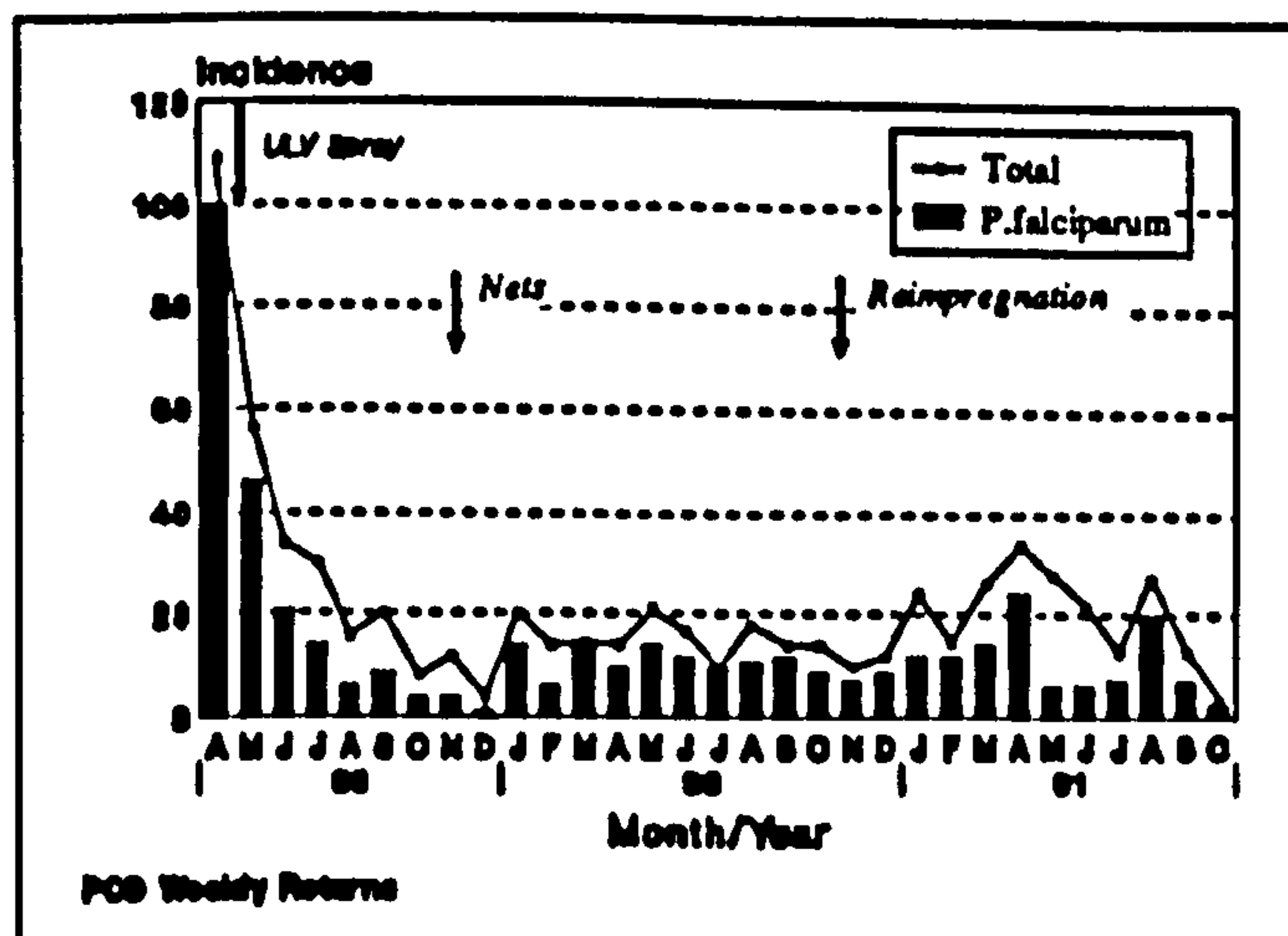
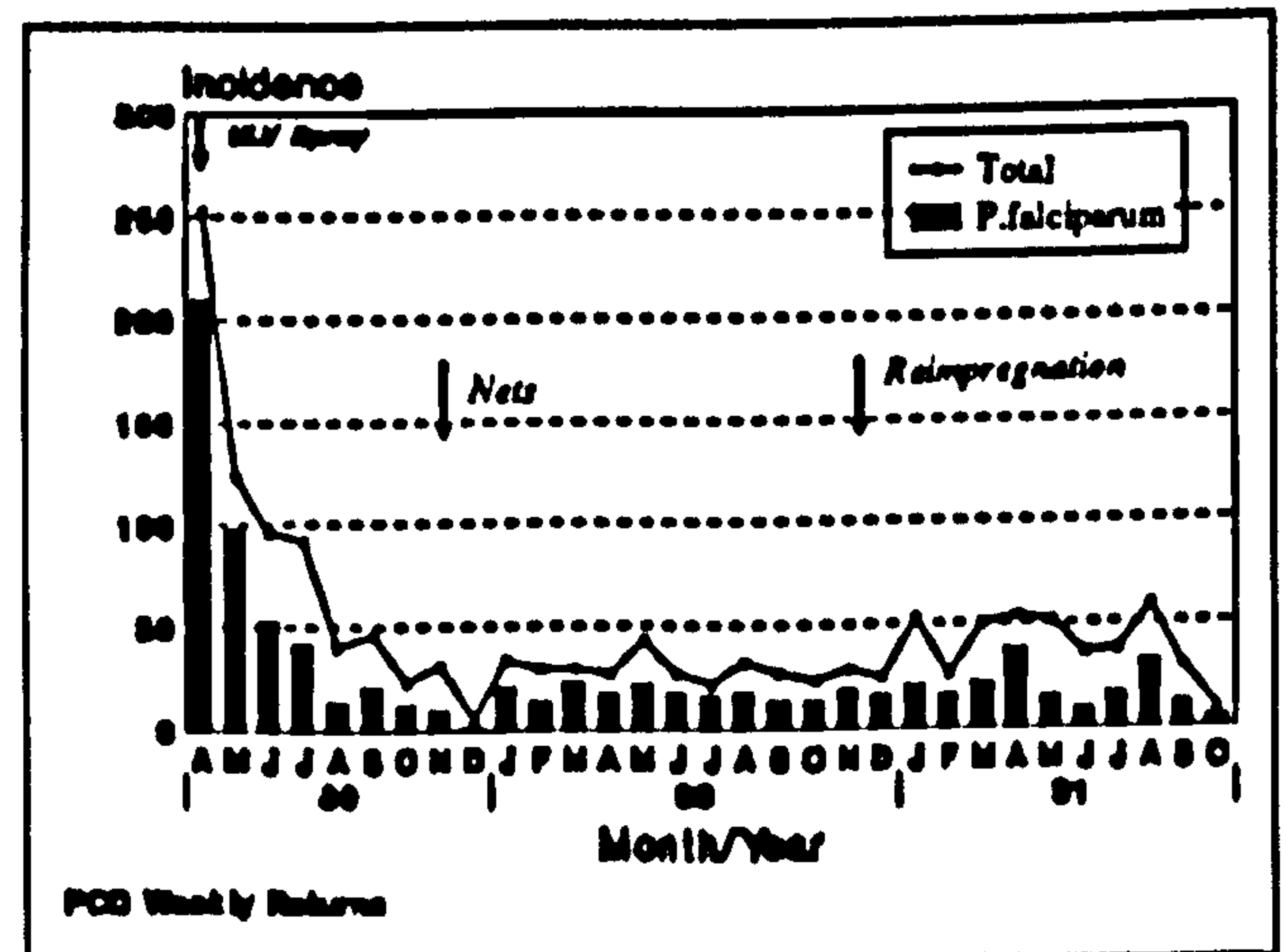


Fig.54: Incidence of malaria in all ages, Kolobangara.



age groups, was maintained with re-impregnation of the bednets.

The incidence in all ages combined also showed a similar pattern with a further significant reduction with permethrin impregnated bednets. The low incidence was maintained with re-impregnations. The cases that occurred were mainly in older age groups, and with *P. vivax* malaria. In this trial area there was an epidemic of *P. falciparum* malaria before bednets were introduced, which was effectively brought down by remedial measures and further maintained with impregnated bednets.

The incidence in infants and 1-4 year old in the DDT area (Vona Vona) are shown in

Fig.55 and Fig.56. In infants any positive effect of DDT was unsustained and inapparent. The incidence had peaks during the first quarter of each year, with a slight rise in total P.falciparum.

Fig.55: *Incidence of malaria in infants, Vona Vona.*

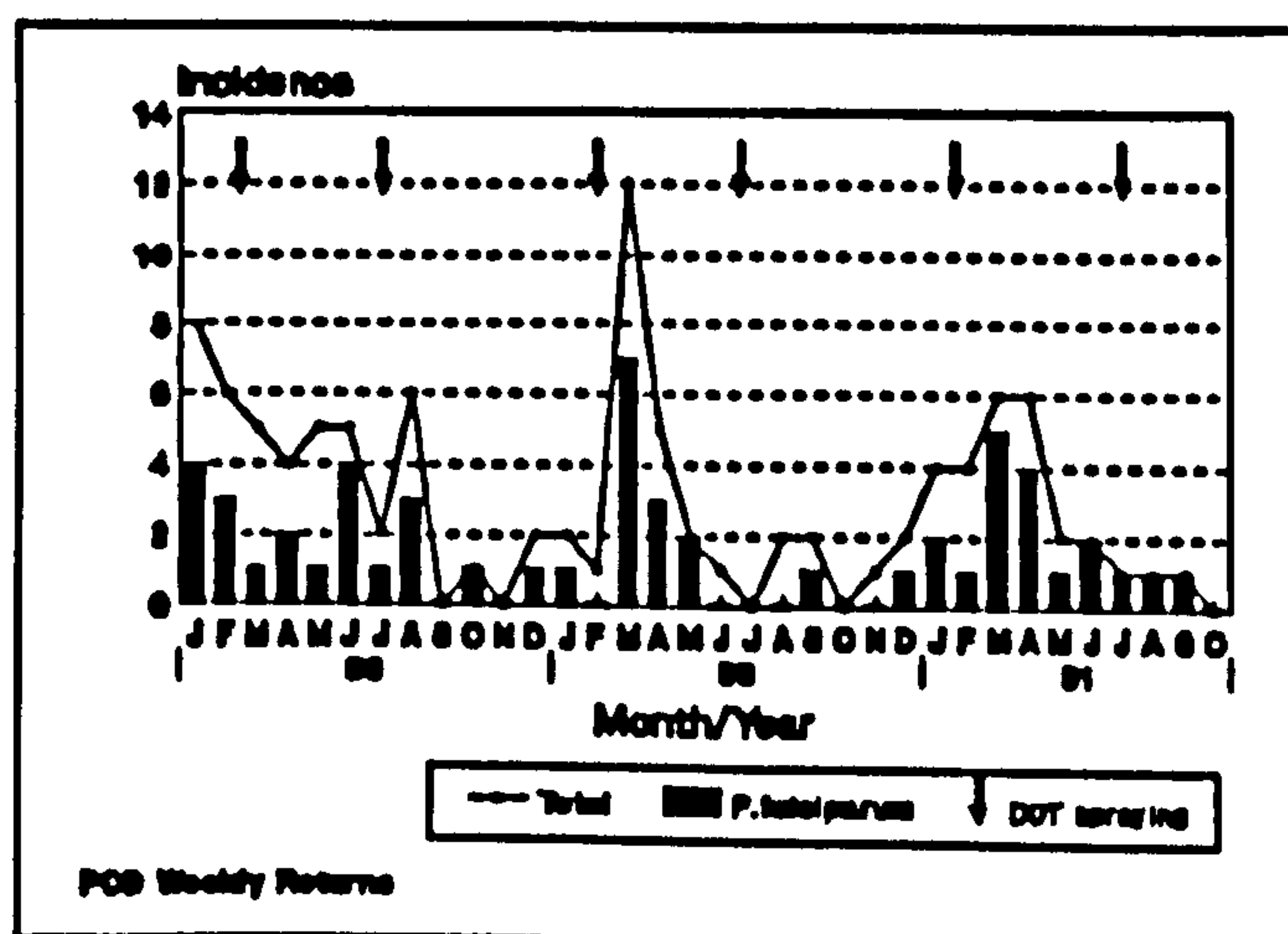
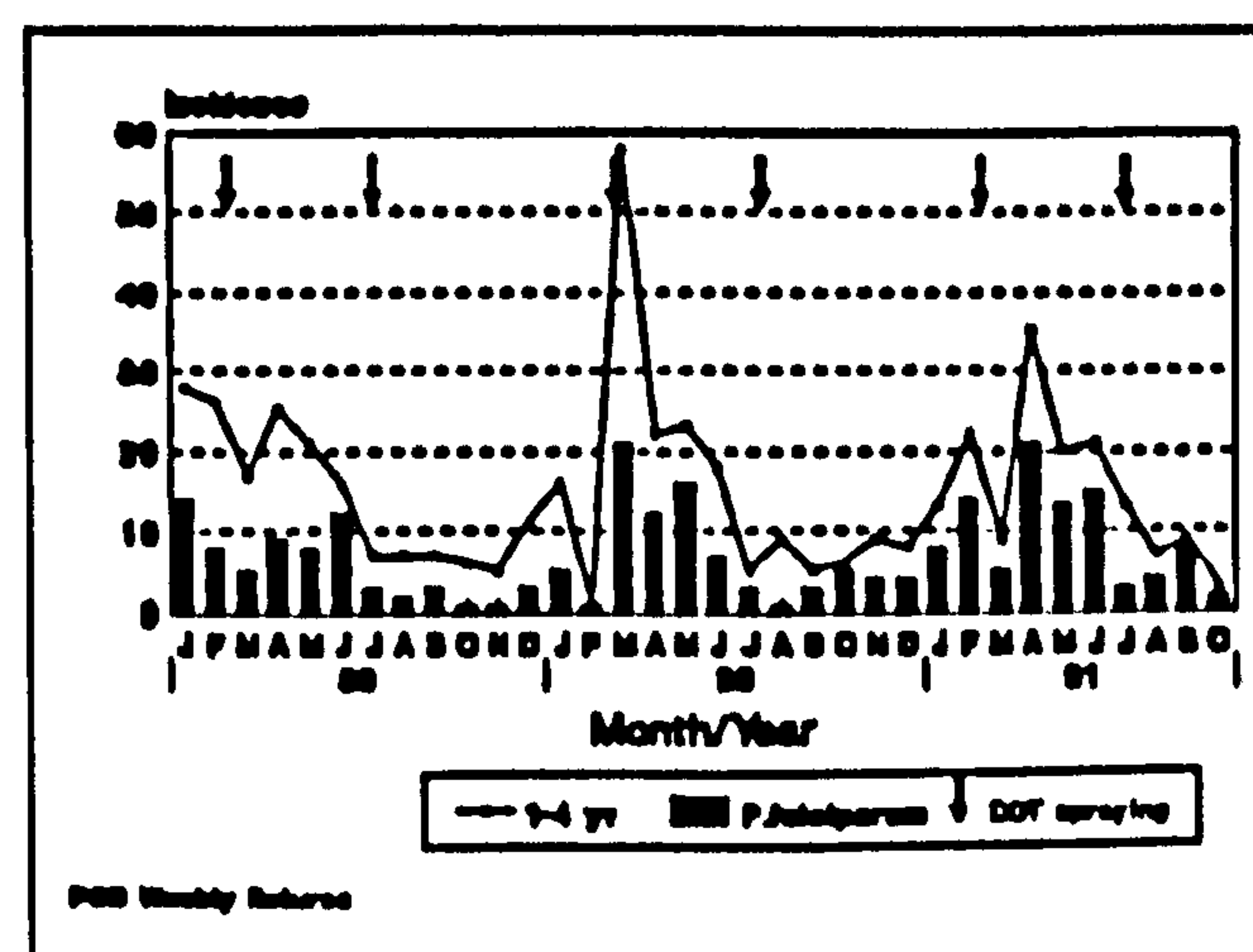


Fig.56: *Incidence of malaria in 1-4 yr old, Vona Vona*



It may be that this is the minimum protection DDT spraying was able to offer infants against malaria in this area with its prevailing conditions. In the 1-4 year old the pattern is similar but with more cases diagnosed from 1990, especially outbreaks of P.vivax, as in infants. There was a gradual increase of P.falciparum cases.

The changes in incidence of the 5-9 year and 10-14 year old in this DDT area are presented in Fig.57 and Fig.58. The pattern in both age groups was similar to that of the 1-4 year old, but with more cases diagnosed in both age specific groups. The outbreaks with P.vivax in the first quarter of 1990 and repeated in the next high transmission season, was obvious in these groups. DDT did not reduce incidence further in these age groups and even though there were some slight increases, there were no marked difference between the monthly incidence between 1989, 1990 and 1991.

The incidence in the 15 years and older, and in all ages combined in Vona Vona are in Fig.59 and Fig.60. In the adults, 15 years and older the pattern was similar with peaks during high transmission seasons and P.vivax outbreaks. However there was a steady high incidence in 1991 with P.falciparum.



Fig.57: *Incidence of malaria in 5-9 yr old, Vona Vona.*

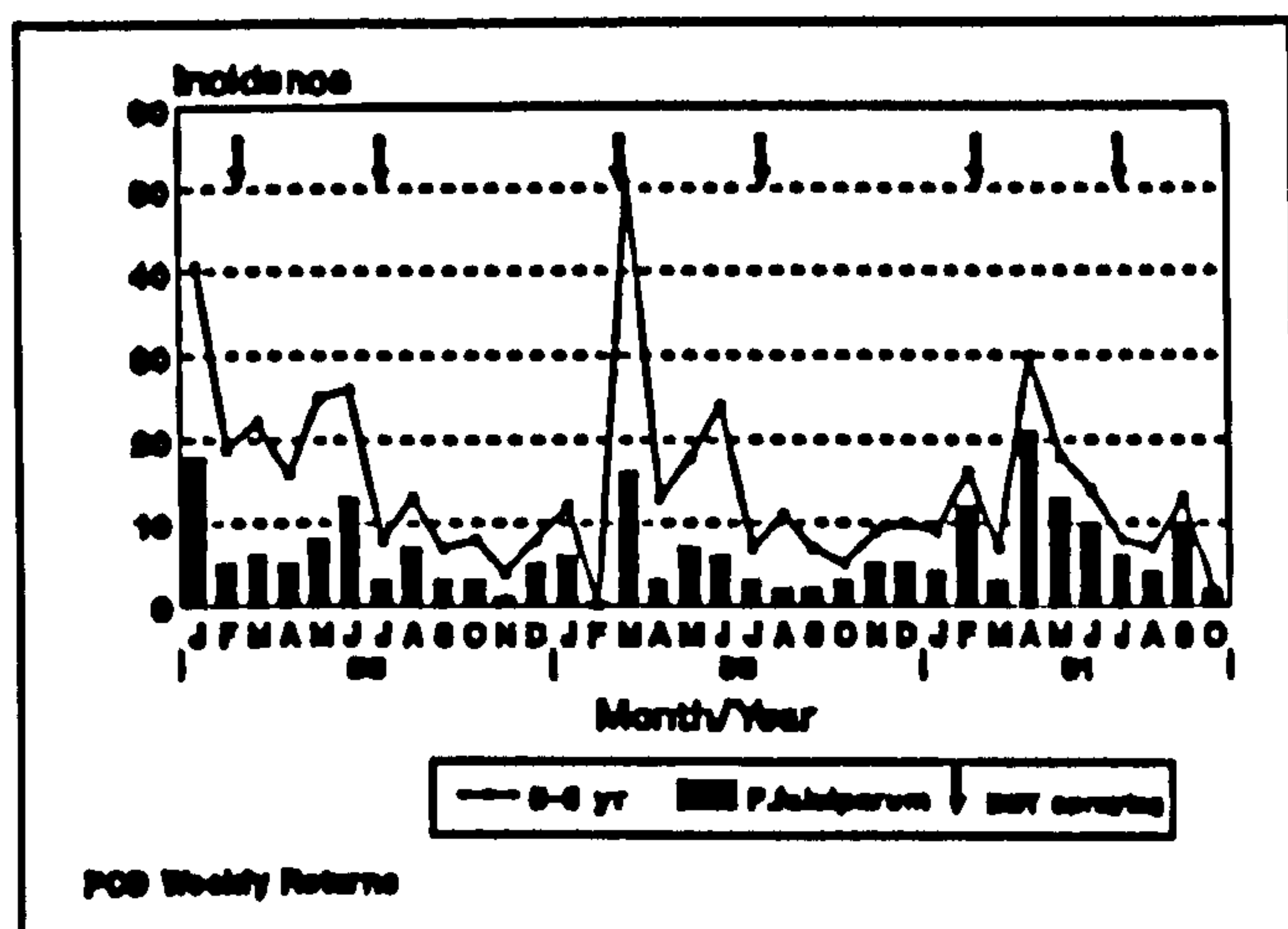


Fig.58: *Incidence of malaria in 10-14 yr old, Vona Vona*

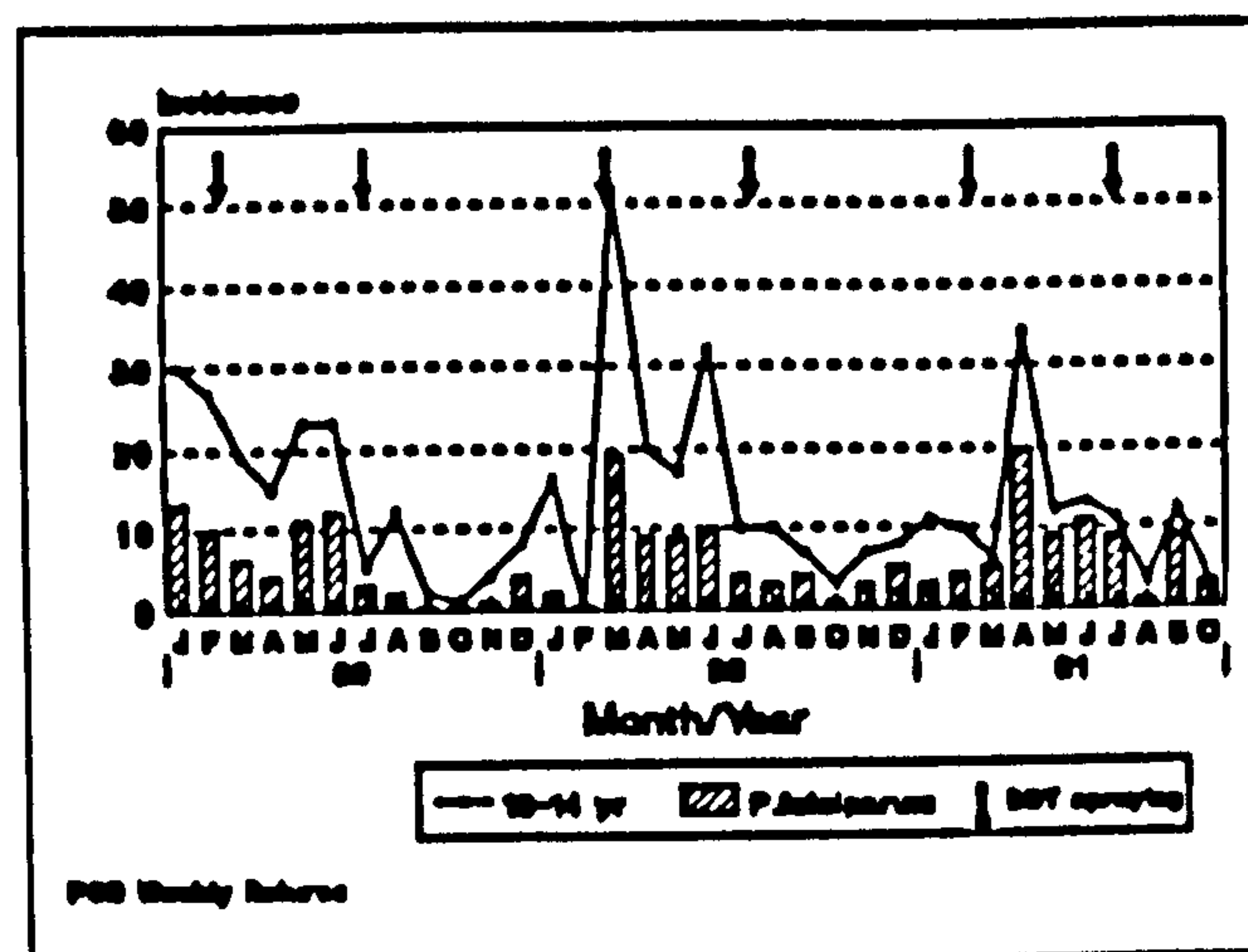


Fig.59: *Incidence of malaria in 15 yr +, Vona Vona*

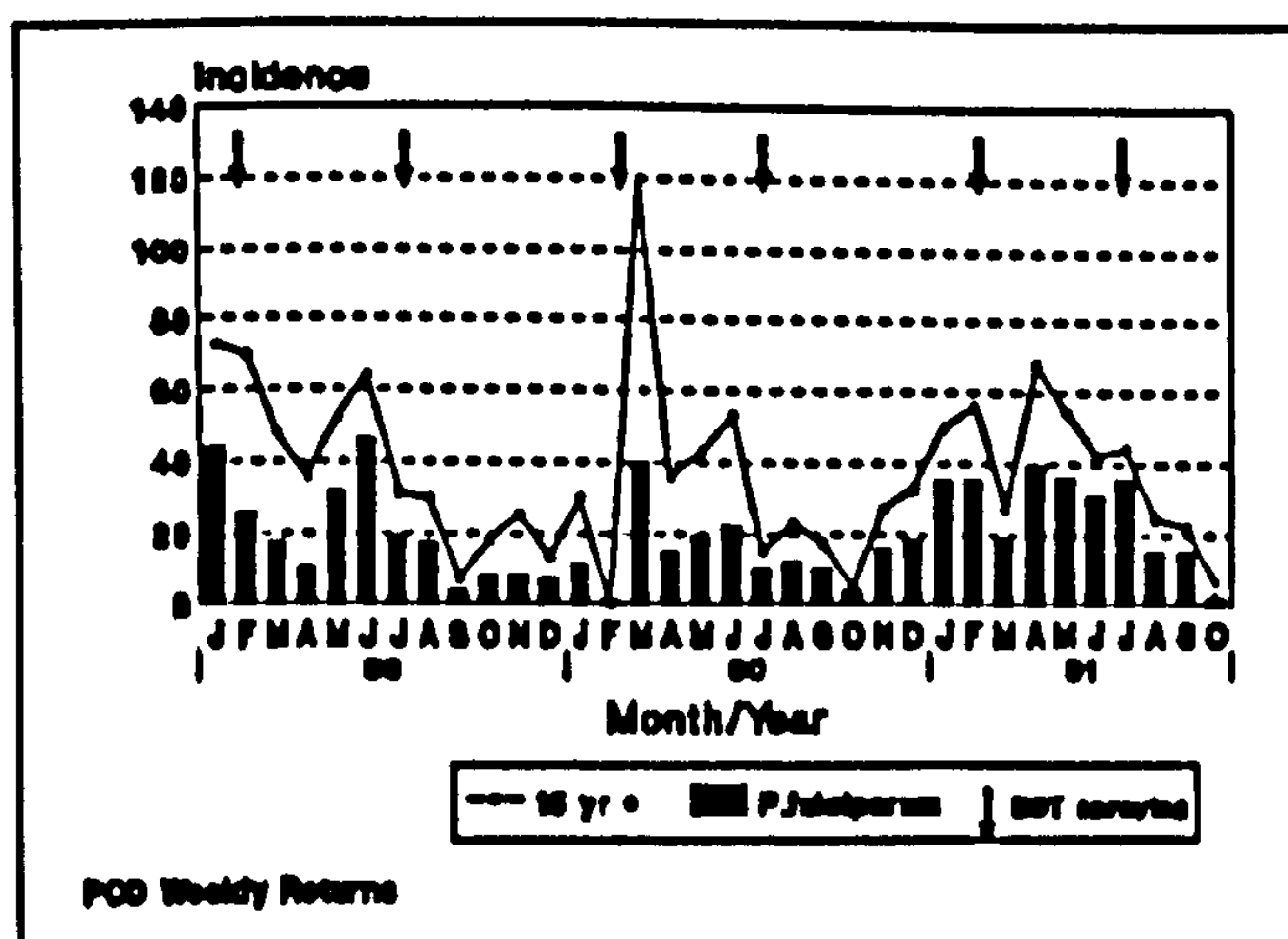
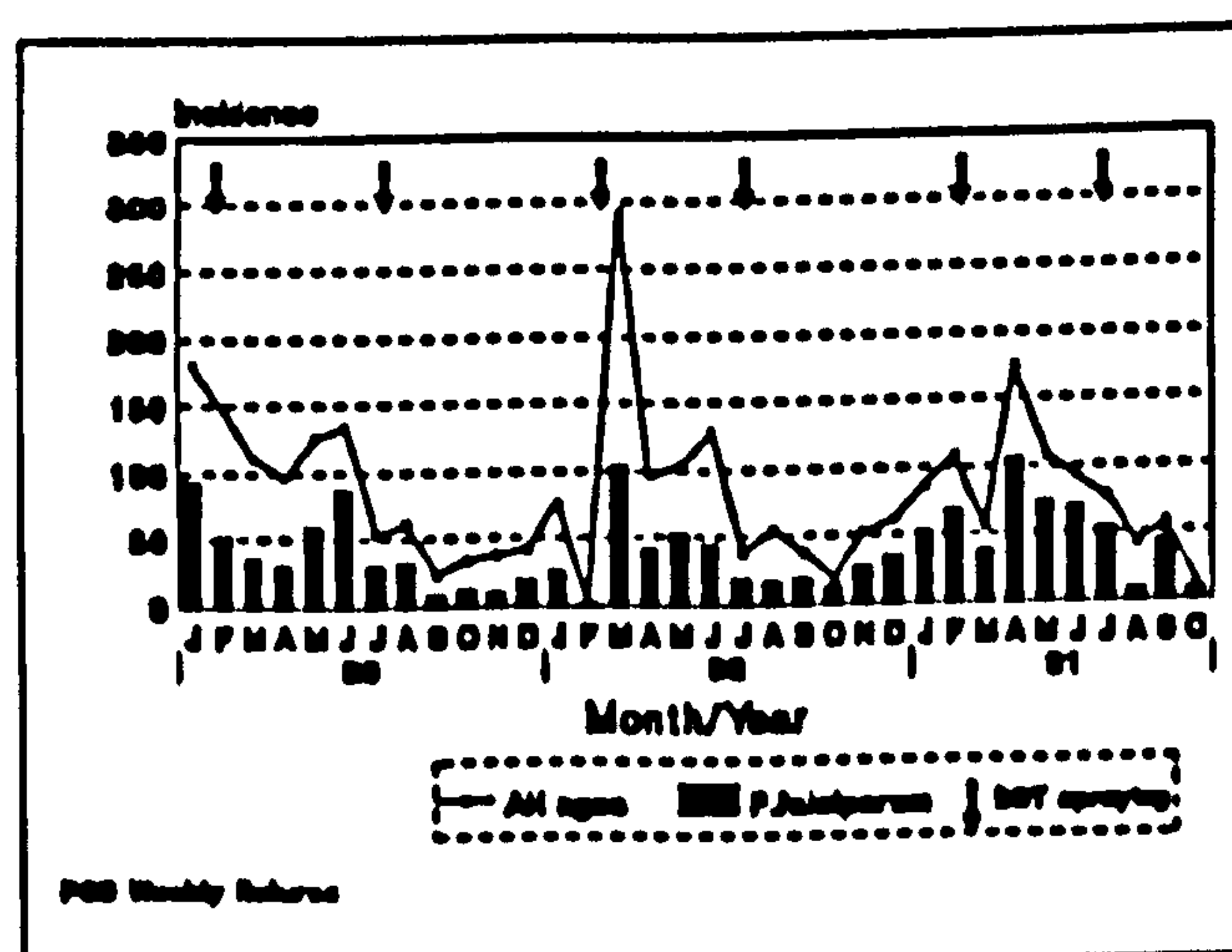


Fig.60: *Incidence of malaria in all ages, Vona Vona.*



When all ages were combined, the incidence followed the same pattern but with increases in 1991, particularly contributed to by older age groups, mainly with P.falciparum infections. There were gradual increases in all ages except infants, and the high level of transmission was sustained despite DDT spraying. It may be that this was the minimal threshold in which DDT could maintain malaria transmission in the situation that prevailed. However with the gradual increase of P.falciparum, whatever effective control there might have been, showed evidence of being lost.



#### 4.3.4 Vella La Vella Island.

The incidence of malaria in infants and 1-4 year old in the Vella La Vella bednet trial are presented in Fig.61 and Fig.62. In infants the incidence dropped and was maintained at a low level especially P.falciparum malaria. In the 1-4 year old the reduction was achieved with

Fig.61: *Incidence of malaria in infants, bednet area in Vella La Vella.*

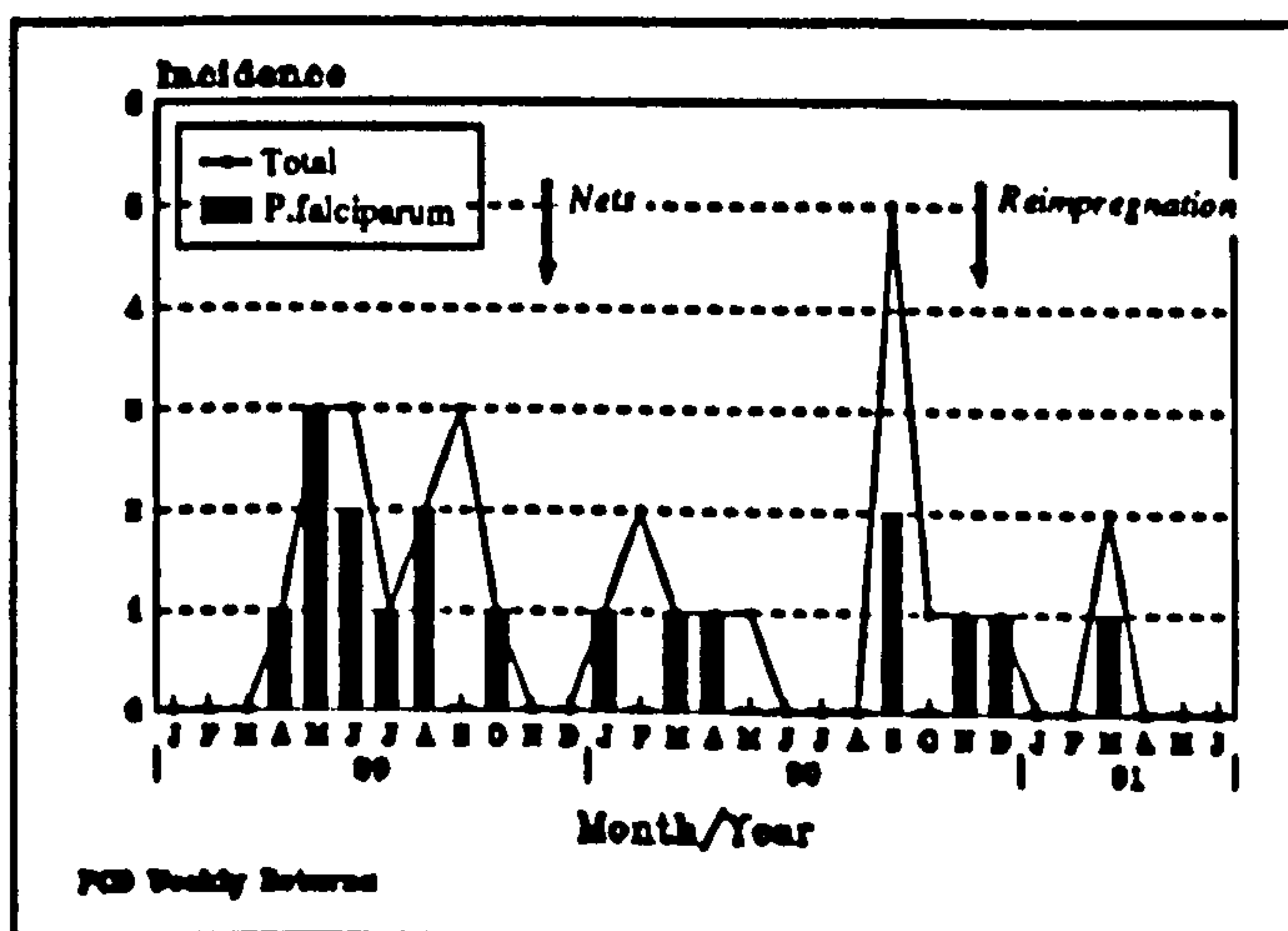
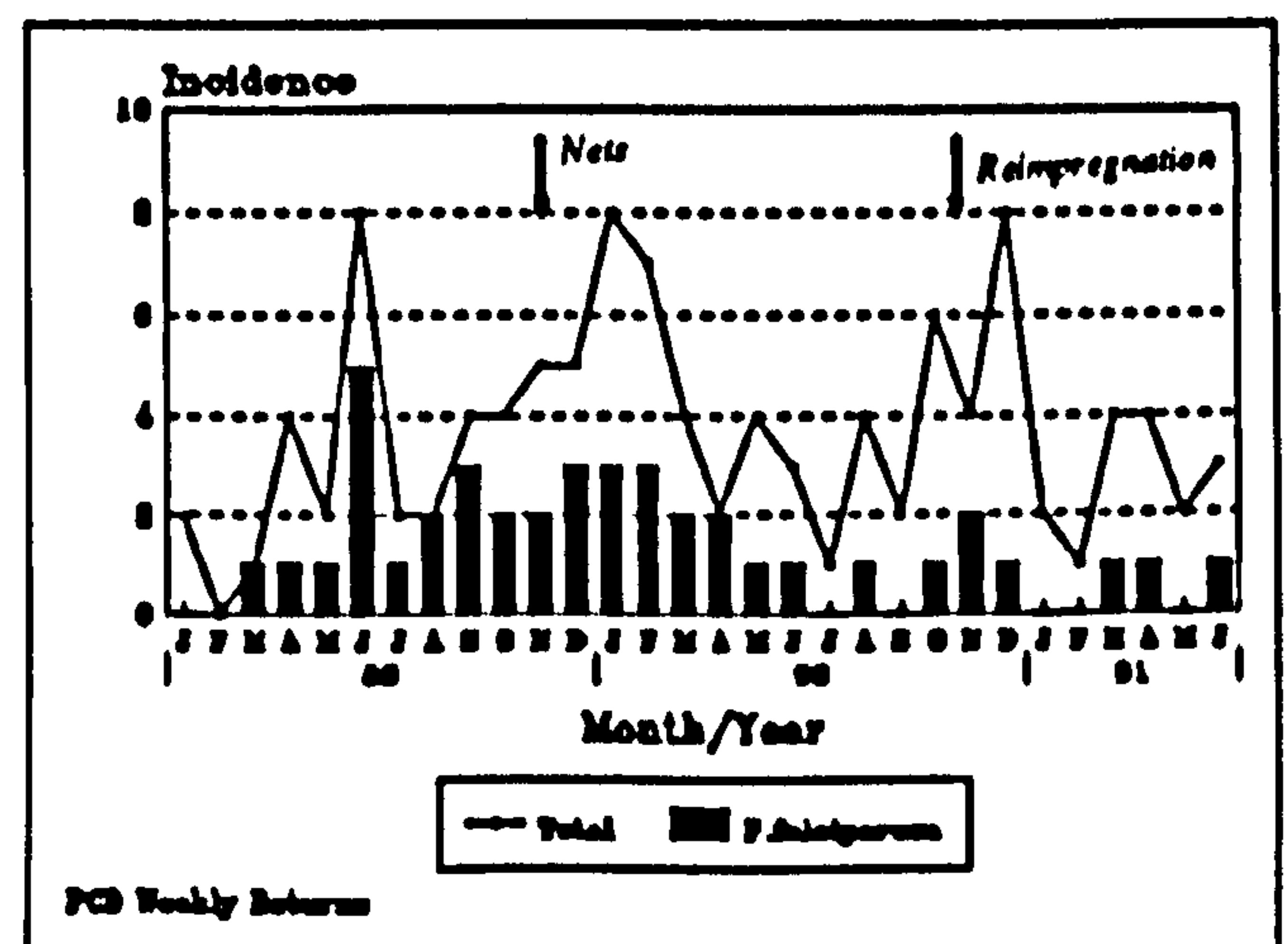


Fig.62: *Incidence of malaria in 1-4 yr old, bednet area, Vella La Vella.*



bednets, especially of P.falciparum which was maintained continuously at a low level. There was a rise with P.vivax, nine months after nets were distributed, but it declined after re-impregnation. Impregnated bednets were especially effective against P.falciparum in this age group.

The results in the 5-9 year and 10-14 year old age groups in the bednet area are in Fig.63 and Fig.64. In the 5-9 year old a low level was achieved and maintained with bednets. A rise nine months after the introduction of bednets, especially with P.vivax, was reduced with re-impregnation. In the 10-14 year old the incidence was reduced and kept low with treated bednets. This low level was maintained throughout with P.falciparum as the main species, except for an outbreak of P.vivax in the first quarter of 1991. The reduction in monthly incidence prior to and after bednets was not as marked even though there was some reduction with P.falciparum.

The incidence in the 15 years and older, and all ages combined, in the bednet area are shown in Figs.65 and 66. The incidence in the 15 year old dropped with bednets and was maintained

Fig.63: *Malaria incidence in 5-9 yr old, bednet area, Vella La Vella*

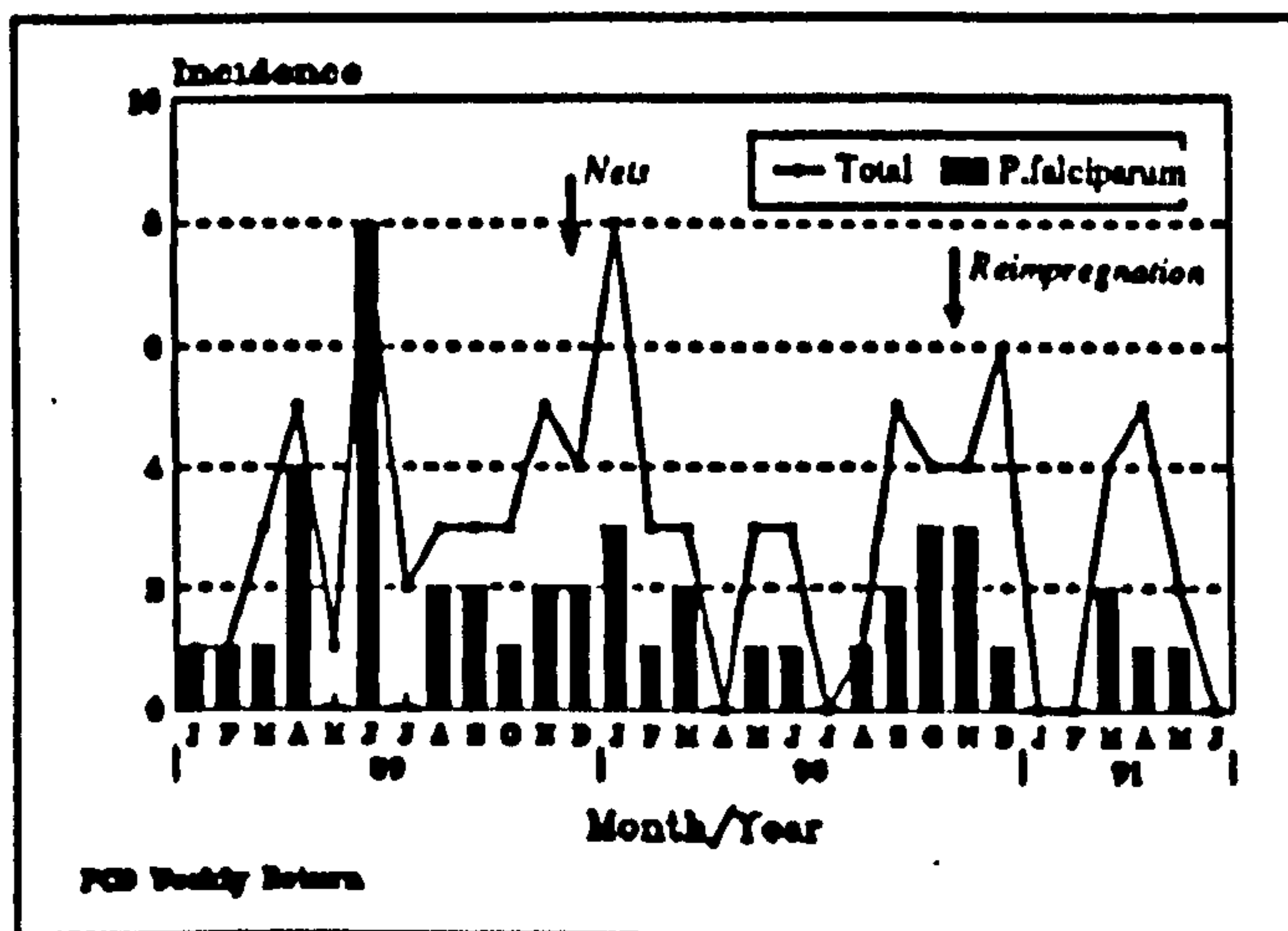
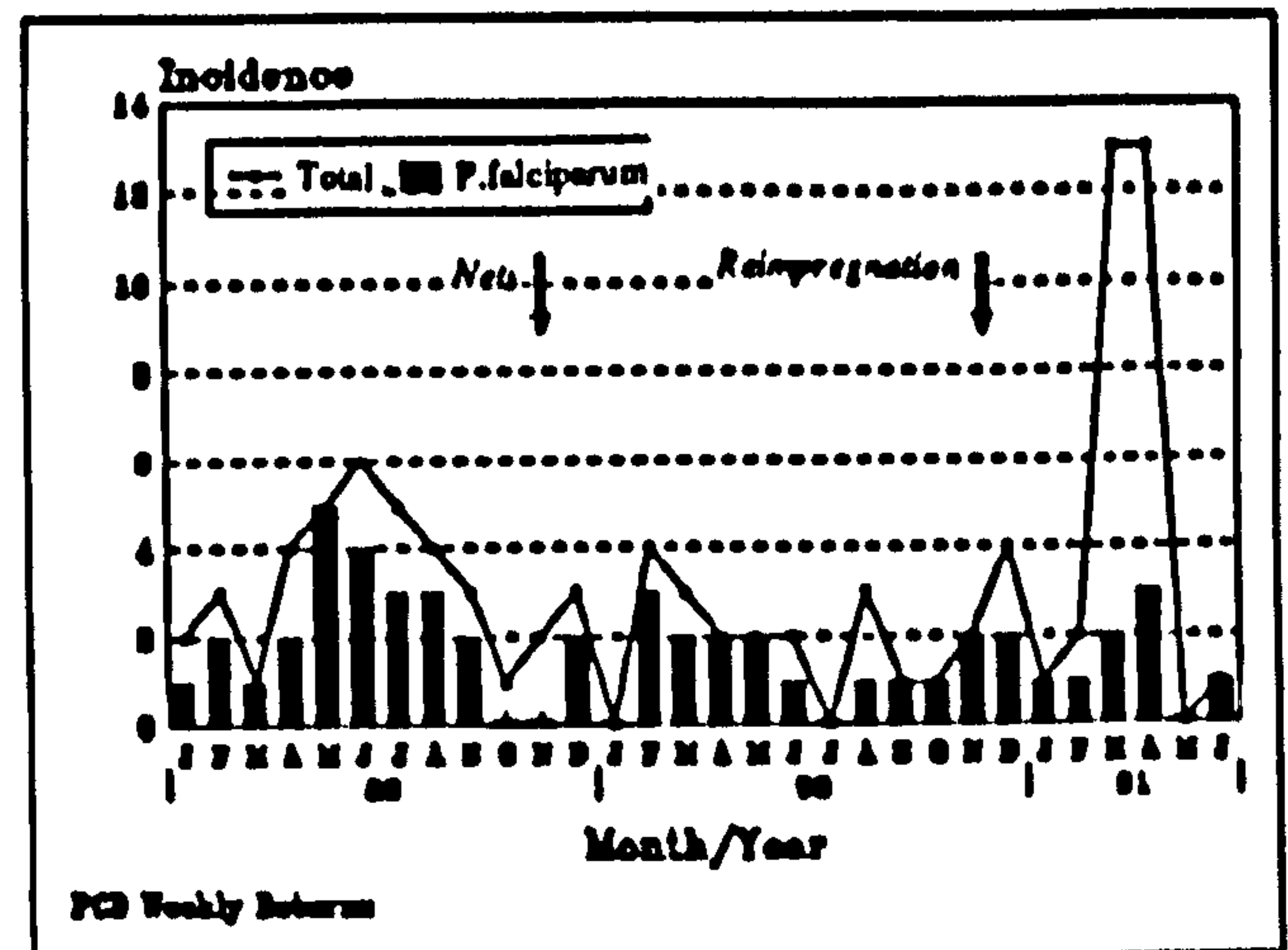


Fig.64: *Malaria incidence in 10-14 yrs old, bednet area, Vella La Vella.*



at a low level. The peaks, mainly with *P.vivax*, during the high transmission season, that were reached prior to the use of bednets did not occur. Infection was mainly with *P.falciparum* except immediately after nets were issued and re-impregnations, including an outbreak of *P.vivax* in the first quarter of 1991. Permethrin impregnated bednets did effectively contain *P.falciparum* at low level in older age groups.

The incidence in all ages combined declined with impregnated bednets, but with peaks during high transmission periods, especially with *P.vivax*. These increases, both with *P.falciparum* and *P.vivax*, were mainly with the older age groups. After a period of control in the seventies and early eighties malaria increased in this susceptible population in the late eighties. The older age groups, contributed to the increases as they were more exposed. Permethrin impregnated bednets effectively curtailed the increasing incidence, in particular with *P.falciparum*, and especially with younger children and kept the incidence at a very low level. The results in infants and 1-4 year olds in the DDT area are shown in Fig.67 and Fig.68. The incidence in infants was maintained at a low level till 1990 when cases started to increase, especially with *P.vivax* when more cases were detected in 1991. The control that was achieved in the eighties was not maintained in the early nineties. In the 1-4 year olds the trend was



Fig.65: *Malaria incidence in 15 yrs + bednet area, Vella La Vella.*

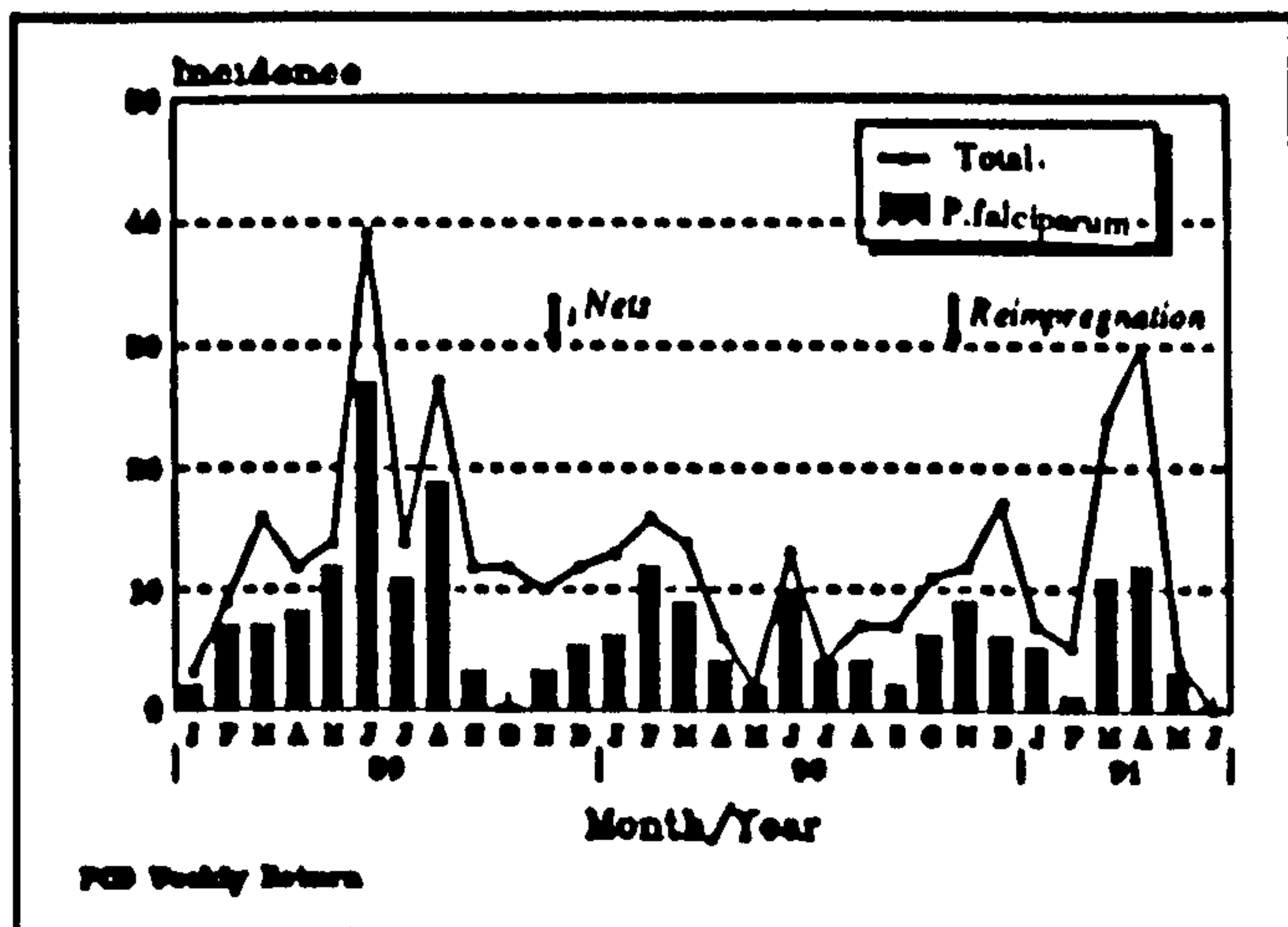
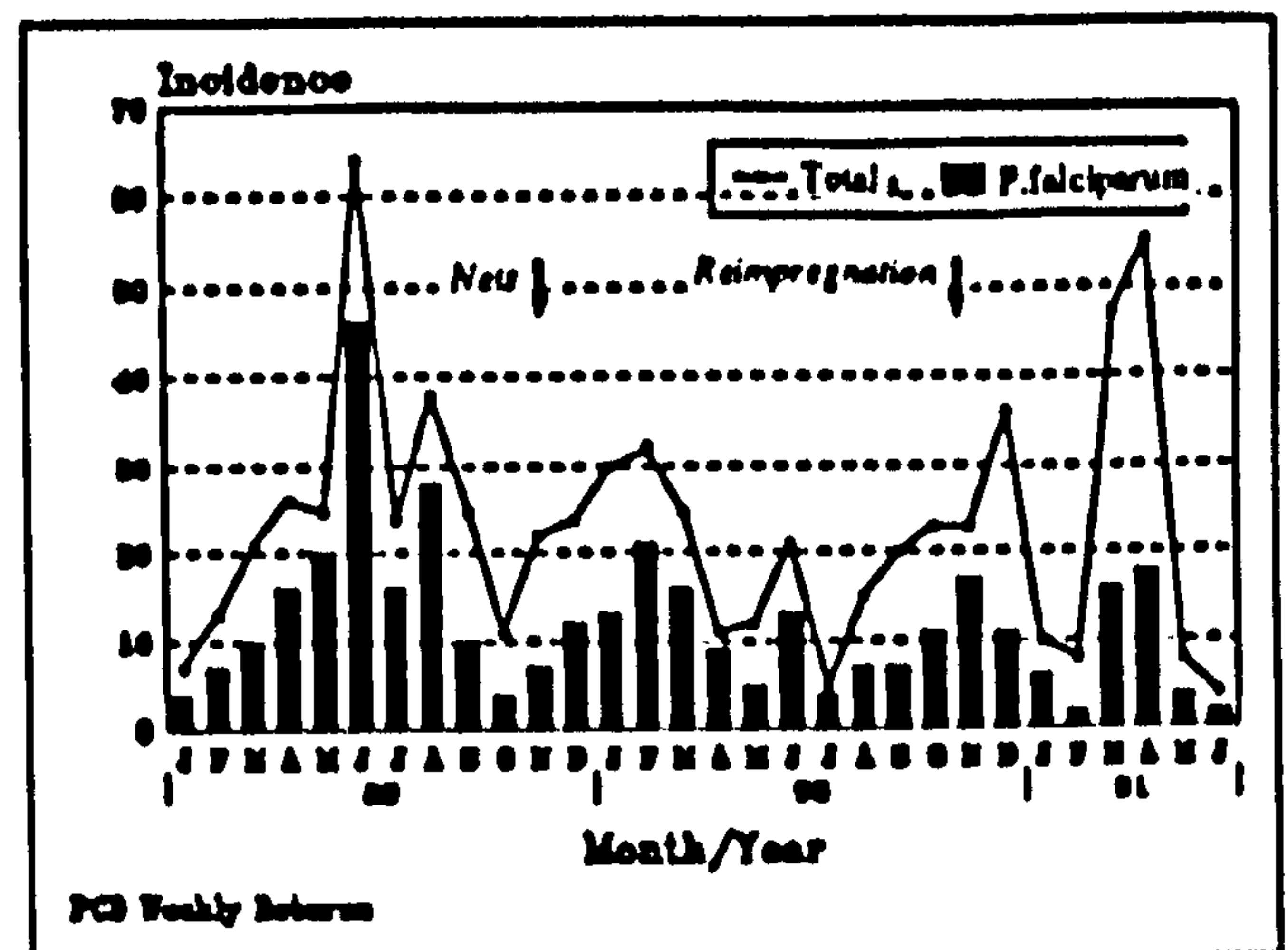


Fig.66: *Malaria incidence in all ages, bednet area, Vella La Vella.*



similar, except that more cases were diagnosed. After a sustained low level the increase was seen in the first quarter of 1990, but much more marked in 1991. Any effective control by DDT spraying was lost. This increase in incidence was particularly with P. falciparum.

Fig.67: *Malaria incidence in infants, DDT area, Vella La Vella.*

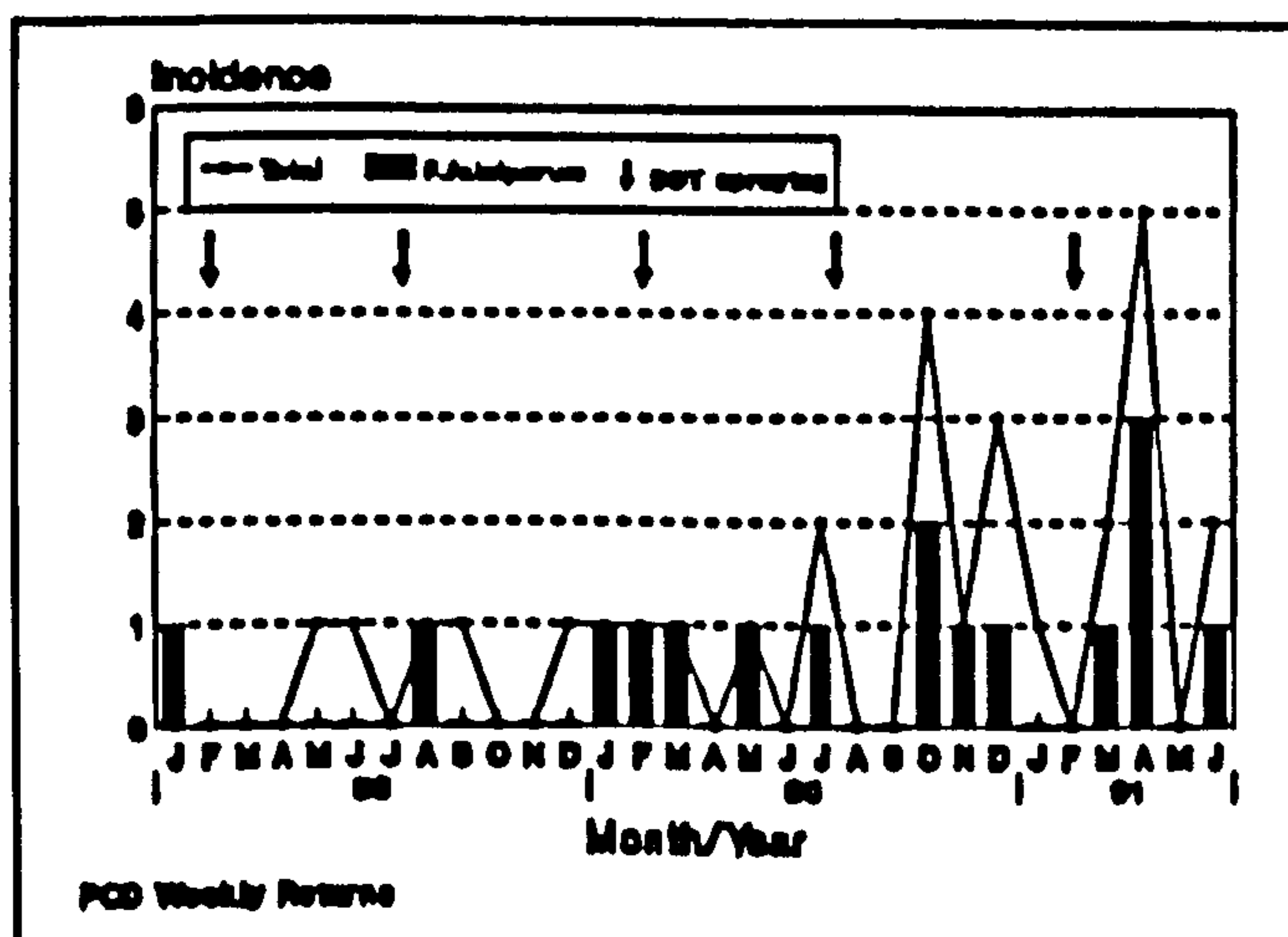
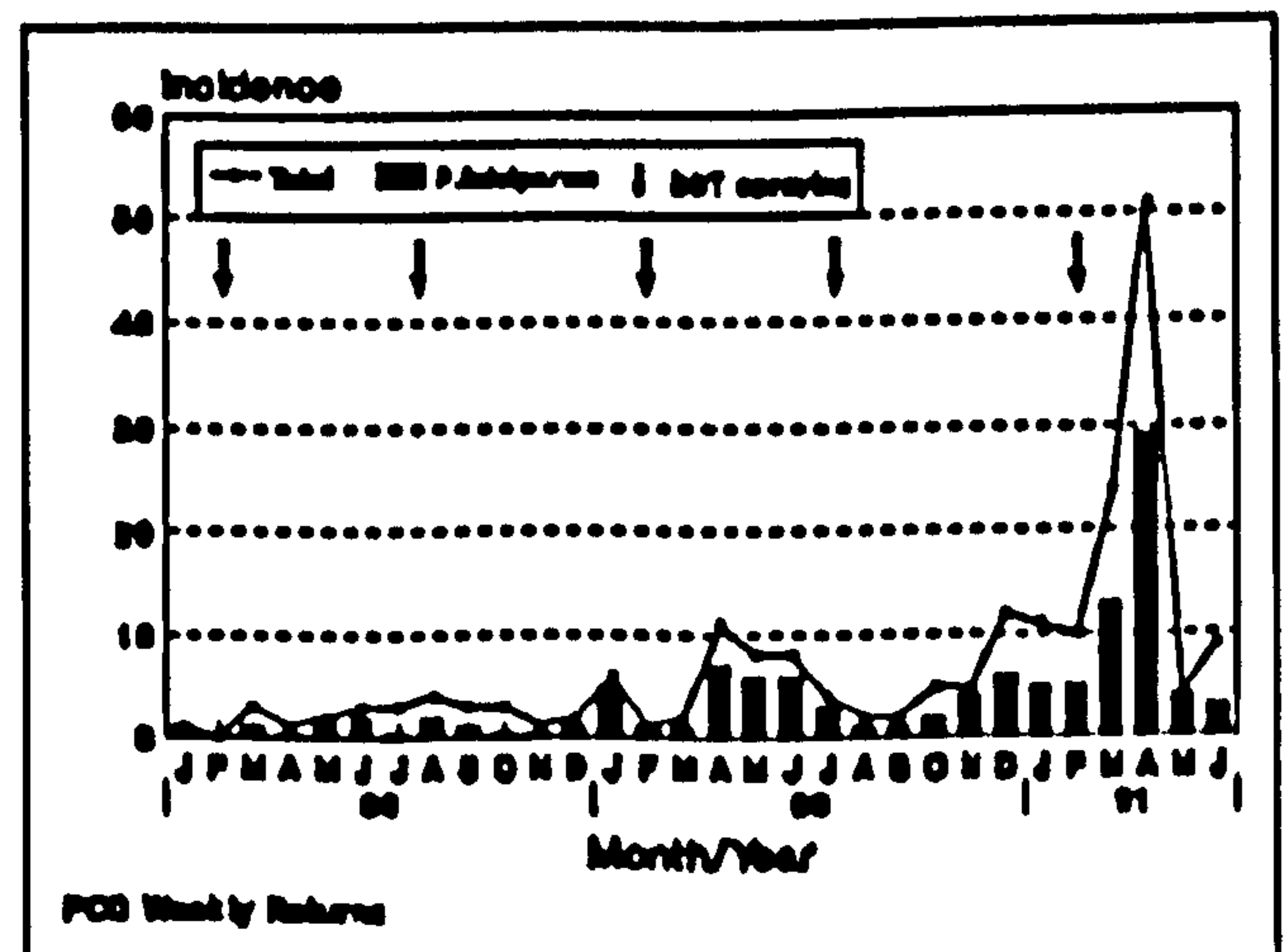


Fig.68: *Malaria incidence in 1-4 yr old, DDT area, Vella La Vella.*



The incidence of malaria in the 5-9 year and 10-14 year old in this DDT area are presented in Fig.69 and Fig.70. The trend in the 5-9 year old was similar with 1-4 year old, with a slight rise in the first quarter of 1990, but a much more marked rise in 1991. This was also similar with the 10-14 year old. These increases were mainly with P. falciparum.



Fig.69: *Malaria incidence in 5-9 year old, DDT area, Vella La Vella.*

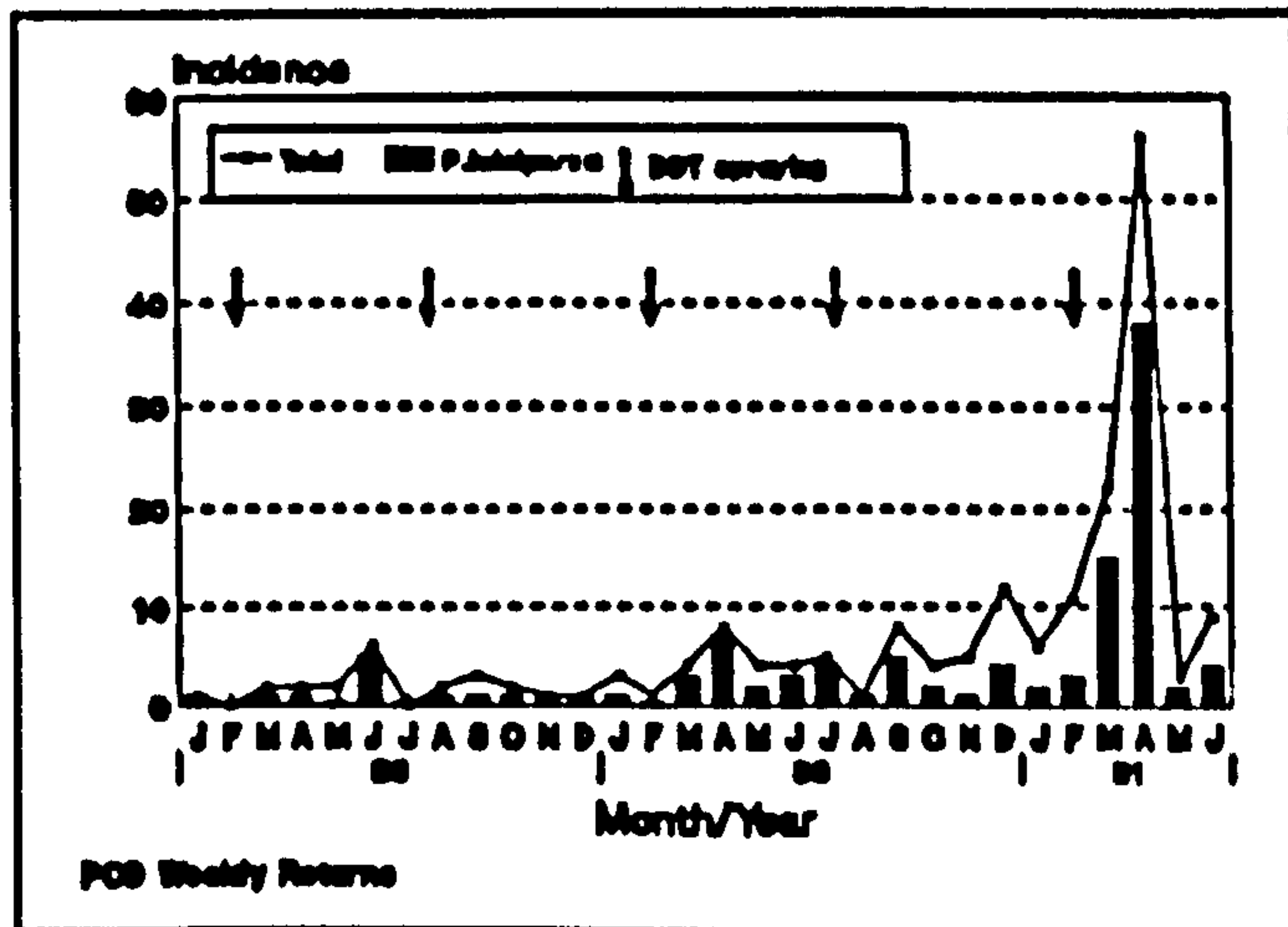
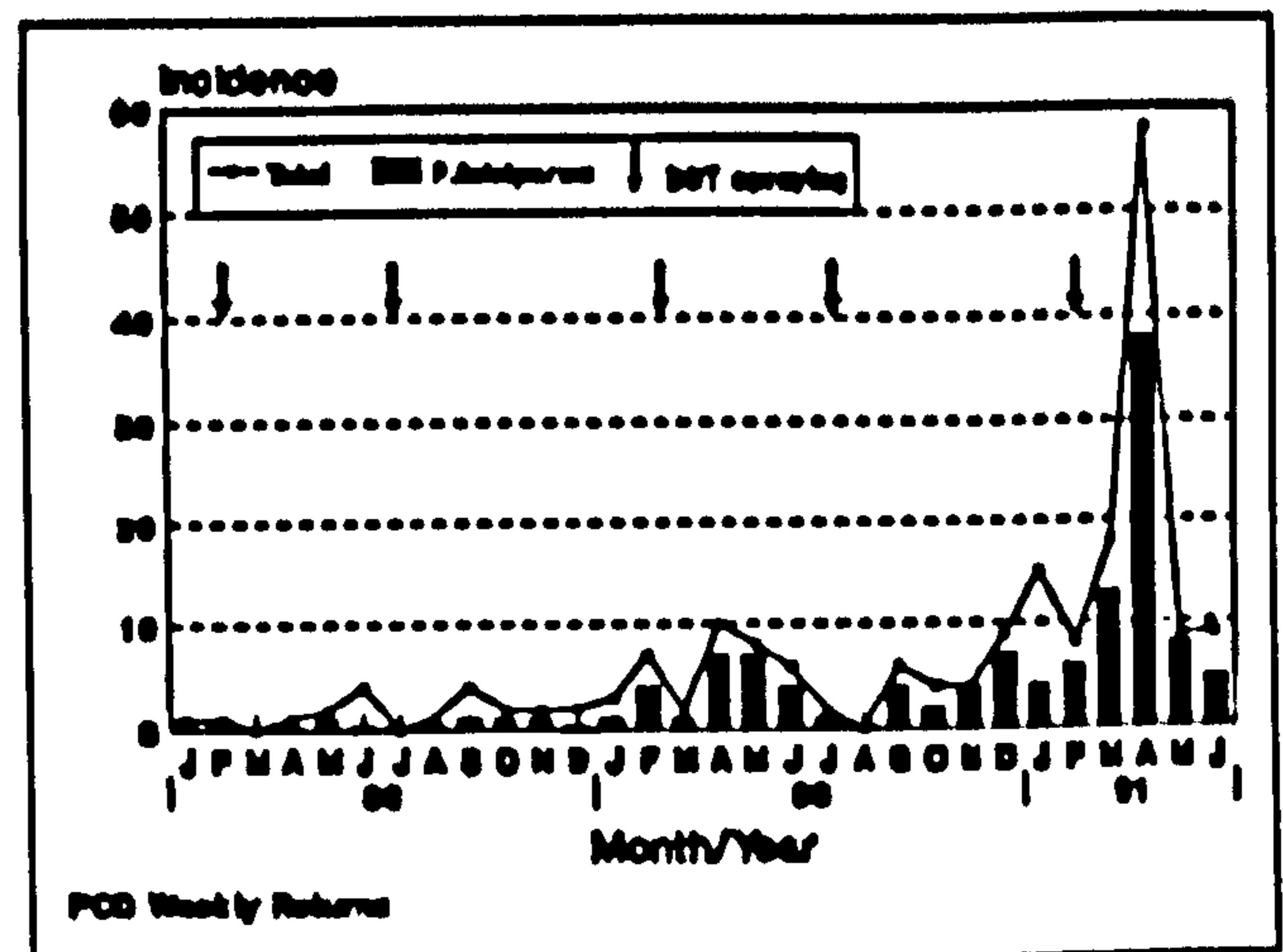


Fig.70: *Malaria incidence in 10-14 yr old, DDT area, Vella La Vella.*



The malaria incidence in the 15 years and older and all ages combined are presented in Fig.71 and Fig.72. The incidence pattern in the 15 years and older was similar to that of the previous age groups. The control that was sustained up to 1989 was not maintained and after a slight rise in 1990, the increase in 1991 was in epidemic proportions especially with

Fig.71: *Malaria incidence in the 15 yrs +, DDT area, Vella La Vella.*

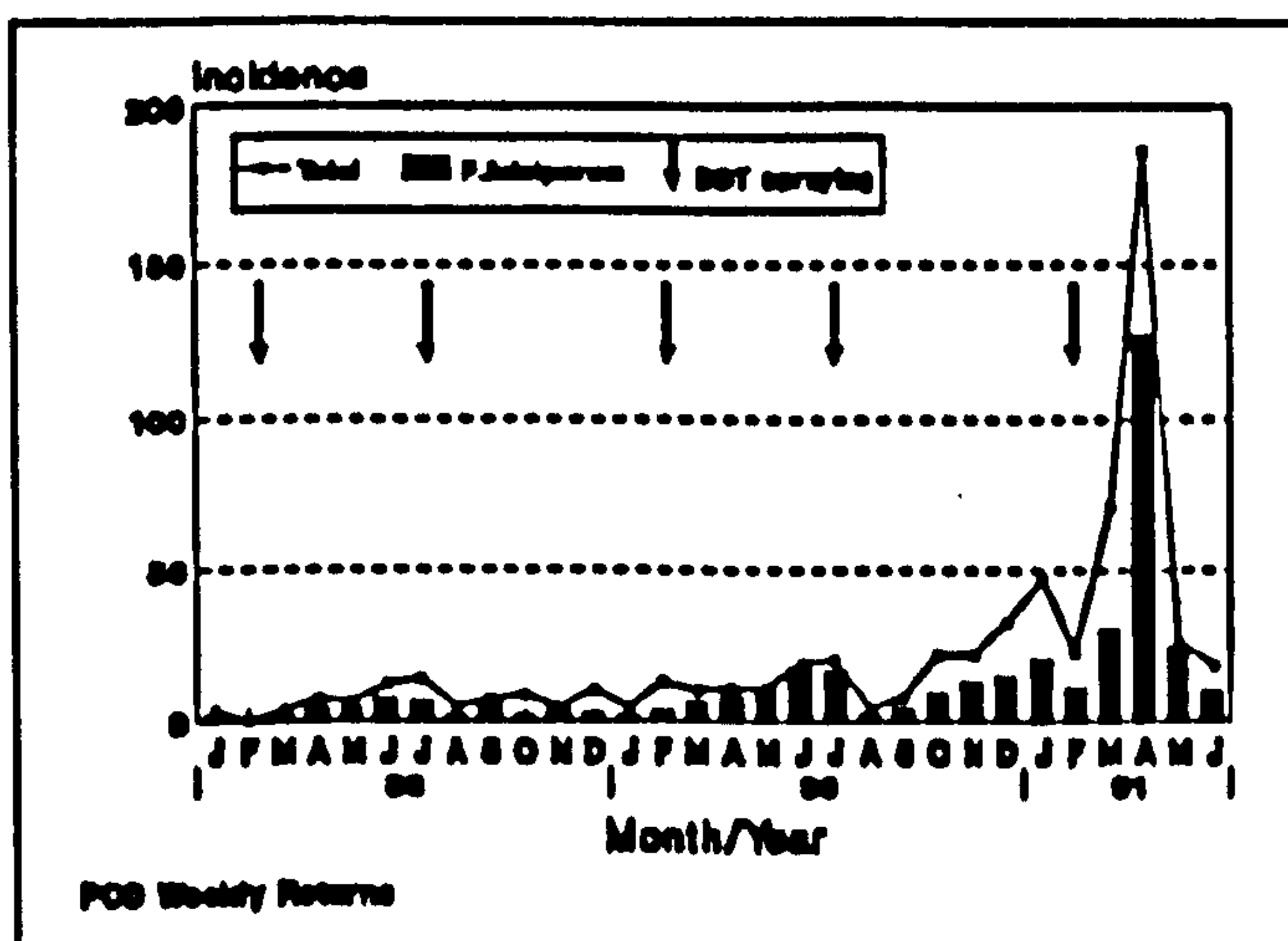
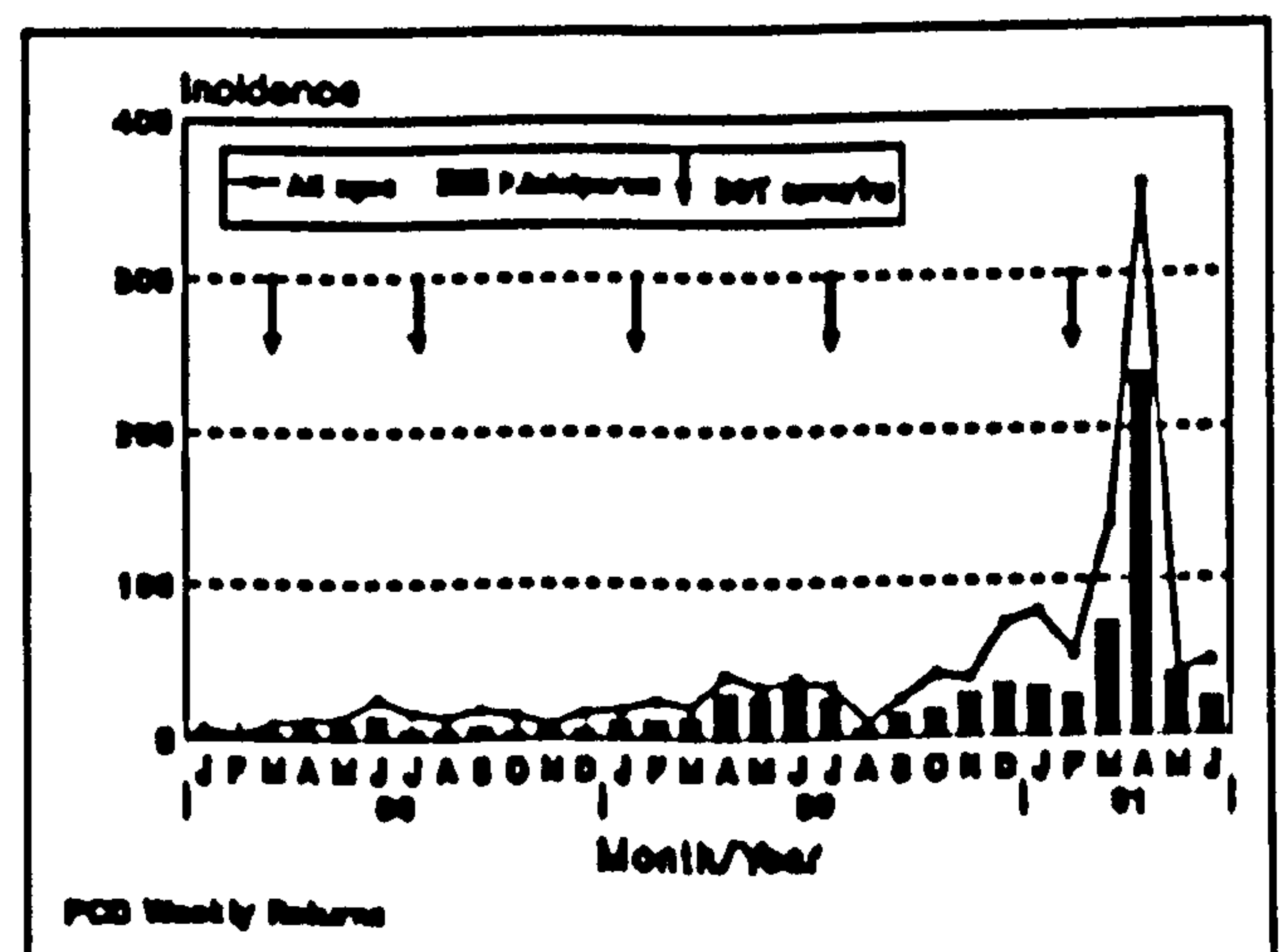


Fig.72: *Malaria incidence in all ages, DDT area, Vella La Vella.*



P.falciparum. DDT spraying no longer maintained the previous effective control of malaria in this area.

The incidence in all ages combined followed the same pattern. After a maintained low

level in 1989, the increase started in 1990 with a slight peak during the high transmission period, but in 1991 the peak was in epidemic proportions, both with P.falciparum and P.vivax. The increase in incidence was in all ages especially the older age groups who were equally susceptible after a long period of low malaria endemicity. The continued DDT spraying did not curtail the outbreak in 1991.

#### 4.3.5 All Bednet Areas Combined.

The available data included a year prior to introduction of bednets in the three trial areas. When the monthly incidence prior to bednets and after introduction of bednets were combined, and the trend of mean monthly incidence determined to show the overall effect of the

**Table 26.** The mean monthly incidence of malaria in infants (per 1000 infants) in the bednet group.

VARIABLE	PRE-BEDNET	POST-BEDNET
Mean	2.73	0.69
Standard deviation	2.12	0.92
Standard error	0.39	0.17
95% Confidence interval of mean	1.97 to 3.49	0.45 to 0.93

**Table 27.** Mean monthly incidence of P.falciparum in infants (per 1000 infants) in the bednet group.

VARIABLES	PRE-BEDNET	POST-BEDNET
Mean	1.77	0.31
Standard deviation	1.55	0.59
Standard error	0.28	0.07
95% Confidence interval of mean	1.22 to 2.32	0.17 to 0.45



intervention, the following results were obtained. The mean monthly incidence in infants prior to bednets and after bednets are presented in Table 26. The reduction with bednets is highly significant, ( $p < 0.0001$ ), especially with P.falciparum as shown in Table 27. The 95% confidential intervals of the respective means, within which the true mean would be found were also different. This resulted in a prevented fraction of malaria incidence in infants to be 74.7%, or 82.5% with P.falciparum.

The mean monthly incidence in the 1-4 year old prior to and after distribution of bednets are in Table 28. The reduction of malaria in this age group is highly significant ( $p < 0.005$ ). This is especially marked with P.falciparum as presented in Table 29.

**Table 28.** Mean monthly malaria incidence in 1-4 year old (per 1000 1-4 yr old) in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNETS
Mean	11.77	5.23
Standard deviation	11.81	2.71
Standard error	2.15	0.34
95% confidence interval of mean	7.56 to 15.98	4.56 to 5.90

**Table 29.** Mean monthly incidence of P.falciparum in the 1-4 year old (per 1000 1-4 yr old) in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNET
Mean	6.97	2.36
Standard deviation	9.40	1.49
Standard error	1.70	0.19
95% confidence interval of mean	3.64 to 10.30	1.99 to 2.73

The respective 95% confidential intervals where their true means would be found were



different especially that of P.falciparum, confirming the real difference caused by permethrin impregnated bednet in this age group. The calculated prevented fraction of malaria incidence in this age group by permethrin impregnated bednets is 55.57%, and that of P.falciparum 66.14%

The mean monthly incidence in the 5-9 year olds prior to and after bednets is presented in Table 30. The reduction in the mean incidence with bednets was highly significant ( $p<0.0001$ ) especially with P.falciparum as presented in Table 31. Their respective 95% confidential intervals in which the true means would lie were different, confirming the significant differences due to bednets, especially with P.falciparum. The calculated prevented fraction

**Table 30.** Mean monthly incidence in the 5-9 year old (per 1000 5-9 yr old) prior to and after bednets in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNETS
Mean	11.57	4.92
Standard deviation	8.74	3.56
Standard error	1.59	0.45
95% Confidence interval of mean	8.45 to 14.69	4.04 to 4.80

**Table 31.** Mean monthly incidence of P.falciparum in the 5-9 year old (per 1000 5-9 yr old) prior to and after bednets in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNETS
Mean	6.30	1.72
Standard deviation	5.71	1.44
Standard error	1.04	0.18
95% Confidence interval of mean	4.26 to 8.36	1.37 to 2.07

of the incidence of malaria with permethrin impregnated bednets in this age group is 57.48%, and that with P.falciparum is 58.14%.

The mean monthly incidence in the 10-14 year old, prior to and after bednets, in the bednets area is presented in Table 32. The effect of impregnated bednets, represented by the reduction in monthly mean incidence is highly significant ( $p < 0.002$ ), especially with P.falciparum as presented in Table 33. Even though the difference is significant, it is less than in the younger age groups as described above.

The respective 95% confidence intervals for both overall mean and P.falciparum mean in this age group, prior to and after bednets, did not overlap, confirming their true differences due to bednets. The calculated prevented fraction malaria incidence by bednets in this age

**Table 32.** Mean monthly malaria incidence in 10-14 year old, (per 1000 10-14 yr old) prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNETS
Mean	12.13	5.28
Standard deviation	11.40	4.45
Standard error	2.07	0.56
95% confidence interval of mean	8.07 to 16.19	4.18 to 6.38

**Table 33.** Mean monthly incidence of P.falciparum in the 10-14 year old (per 1000 10-14 yr old) prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNETS	POST-BEDNETS
Mean	7.20	2.39
Standard deviation	8.23	2.16
Standard error	1.50	0.27
95% Confidence interval of mean	4.26 to 10.14	1.86 to 2.92

group was 56.47%, and that of P.falciparum was 66.81%.

The mean monthly incidence in the 15 years and older is presented in Table 34. The



reduction in means due to bednets is highly significant ( $p < 0.005$ ) even in this older age group.

This is also especially with P.falciparum which is presented in Table 35.

**Table 34.** Mean monthly incidence in the 15 years and older, (per 1000 15 yrs and older) prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNET	POST-BEDNET
Mean	28.03	15.17
Standard deviation	22.37	6.63
Standard error	4.07	0.83
95% Confidence interval of mean	20.05 to 36.01	13.54 to 16.80

**Table 35.** Mean monthly incidence of P.falciparum in the 15 years and older (per 1000 15 yrs and older), prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNET	POST-BEDNET
Mean	18.08	8.06
Standard deviation	19.57	4.92
Standard error	3.56	0.62
95% Confidence interval of mean	11.10 to 25.06	6.84 to 9.28

The standard deviation after bednets, was smaller, indicating less variation due to some reasonable effective control by the intervention. The 95% confidence intervals in this age group after bednets was different, indicating the true difference where the true means would be found. The prevented fraction of malaria incidence in this older age group was 45.88%, and that of P.falciparum was 55.42%.

The mean monthly incidence in all ages, prior to and after bednets, in the bednet group, is as presented in Table 36. The mean reduction in all ages with bednets was quite significant ( $p < 0.001$ ), especially with P.falciparum, as presented in Table 37. The respective 95% confidence



interval with both means and all ages, after bednets, was different from that prior to bednets in this same group. This difference was more marked with P.falciparum, especially in the younger

**Table 36.** Mean monthly malaria incidence in all ages (per 1000 people), prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNET	POST-BEDNET
Mean	66.20	31.39
Standard deviation	53.77	17.13
Standard error	9.78	2.14
95% Confidence Interval of mean	47.03 to 85.37	27.20 to 35.58

**Table 37.** Mean monthly incidence of P.falciparum in all ages (per 1000 people), prior to and after bednets, in the bednet group.

VARIABLE	PRE-BEDNET	POST-BEDNET
Mean	40.40	14.81
Standard deviation	42.46	7.90
Standard error	7.72	0.99
95% Confidence Interval of mean	25.27 to 55.53	12.87 to 16.75

groups. This confirmed their true difference with the intervention. The smaller standard deviation after bednets indicated that this intervention provided some effective control which is sustained and the incidence did not vary much with other external factors. Impregnated bednets produced a reduction in the overall incidence (prevented fraction) of 52.58%, or 63.34% in P.falciparum.

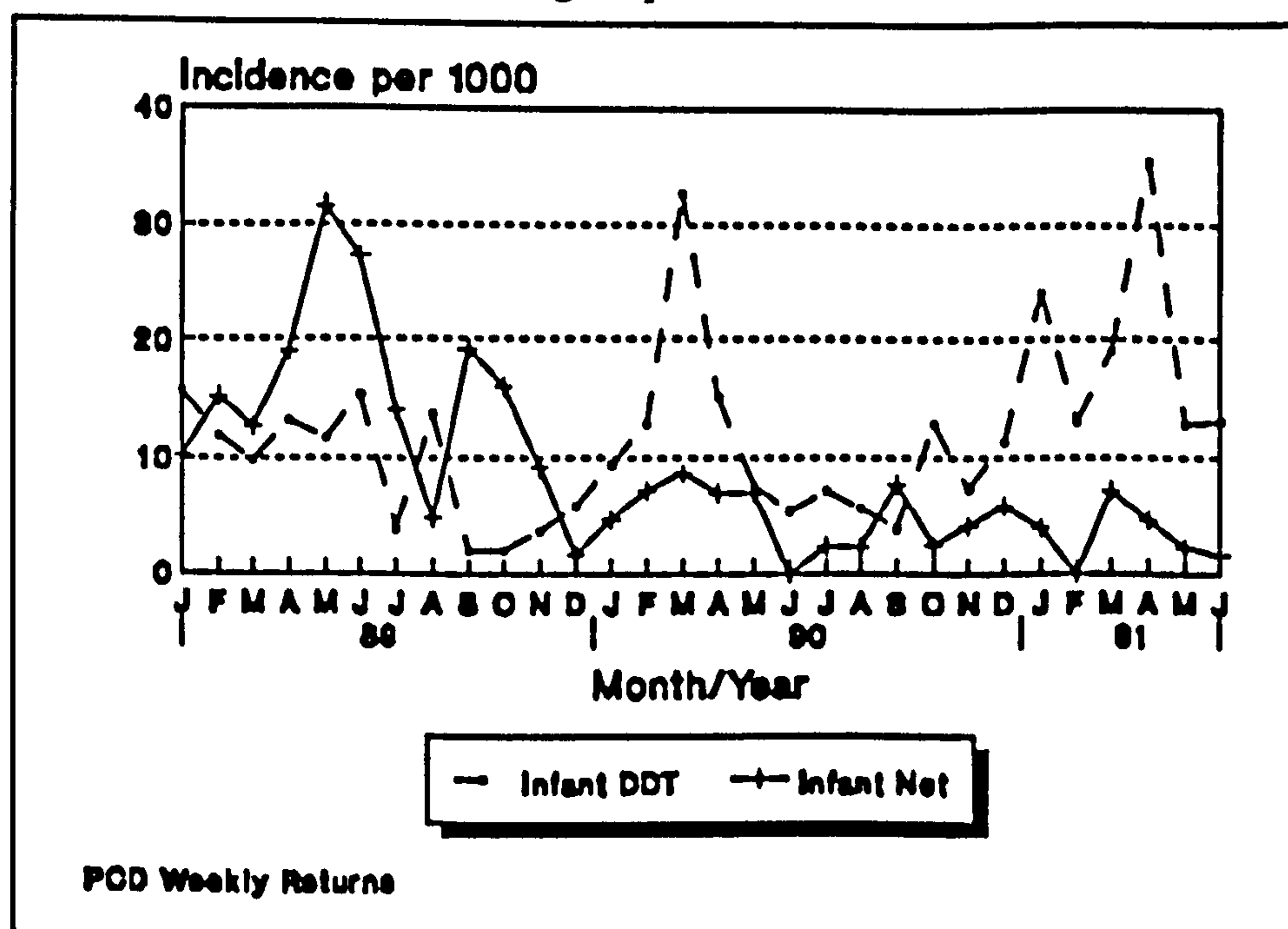
#### 4.3.6 DDT versus Bednets.

When the results in incidence are combined and compared with those of DDT by the age specific groups, the results are as in the following figures. The trend of mean monthly incidence

in infants are shown in Fig.73. Prior to introduction the incidence in the bednet group was higher, with large variations. With bednets the monthly incidence was maintained at about 5 cases per 1000 infants. The incidence in the DDT group however increased and varied enormously with transmission and ended up at the end of the study at about 13 cases per 1000 infants. This meant a disease prevented fraction of 53.33% in infants with bednets over that of DDT.

The mean monthly incidence in the 1-4 year old are in Fig.74. The incidence in the DDT group was initially lower but started to rise in 1990 and fluctuated with seasons and was higher than in the bednet area at a monthly mean of 42.5 cases per 1000 population. The incidence in the bednet area was initially high and varied enormously prior to bednet introduction, but was reduced and maintained at a mean low monthly incidence of about 10.1 cases per 1000

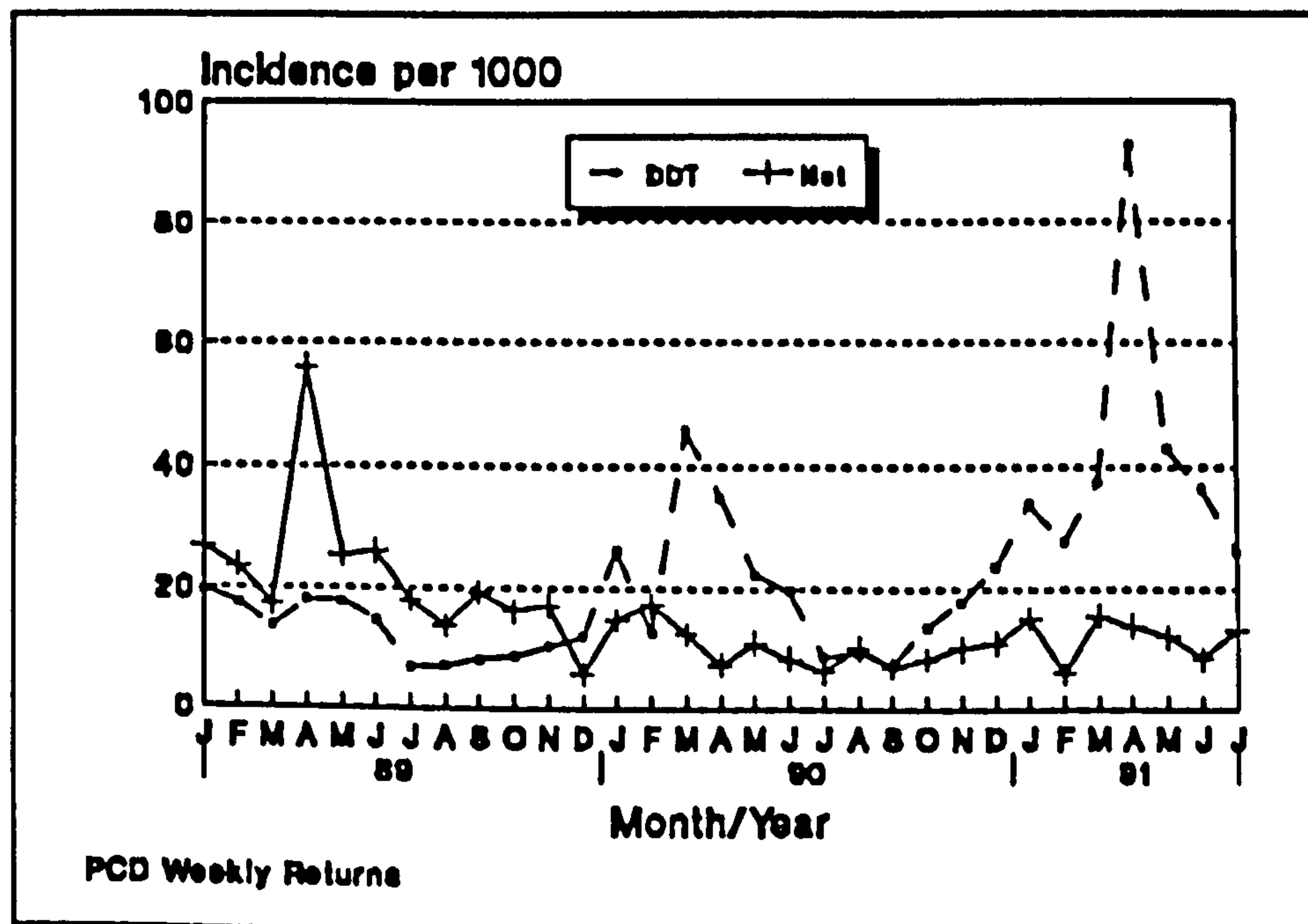
**Fig.73:** *The mean monthly malaria incidence in infants in intervention groups.*



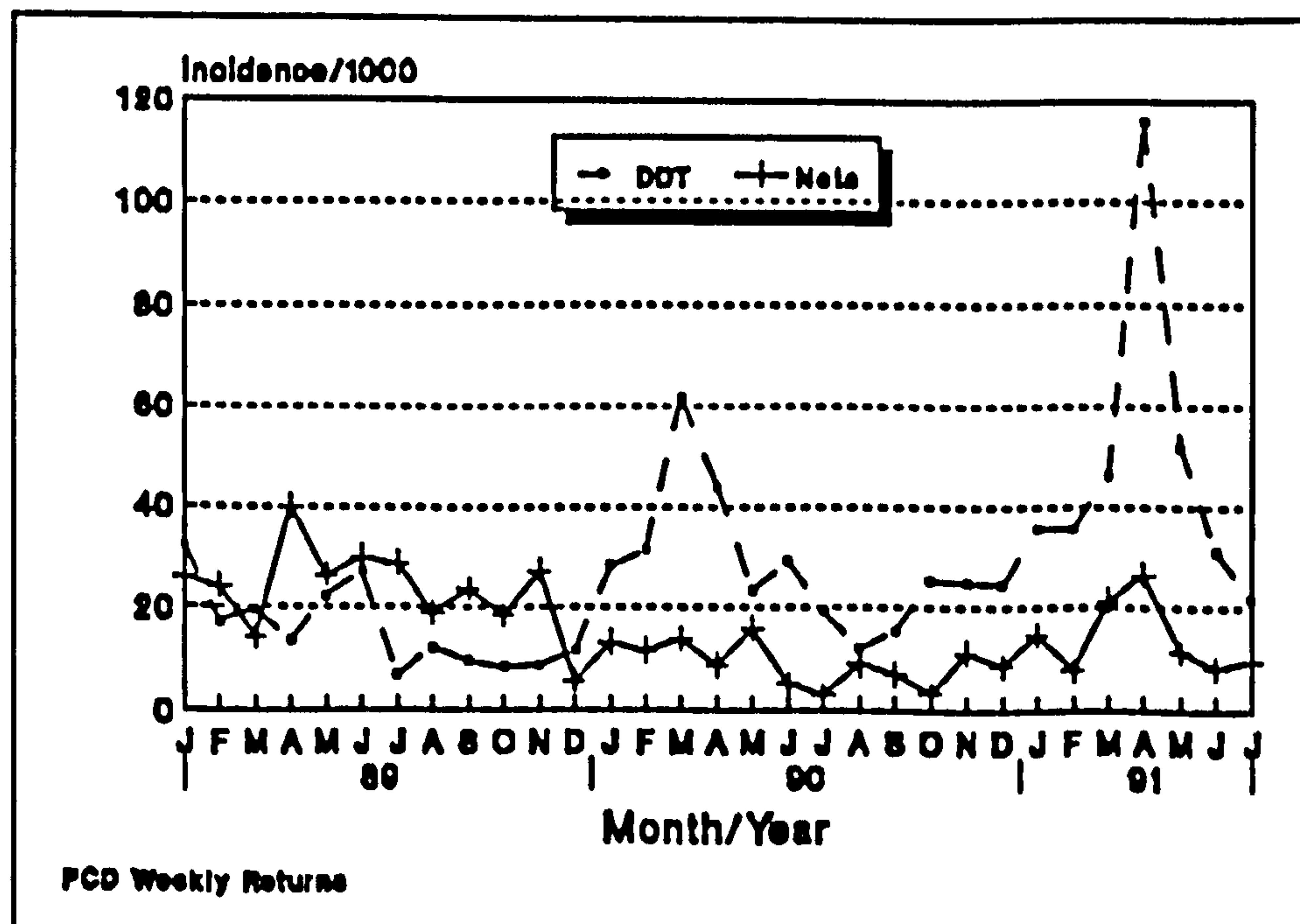
population. Permethrin impregnated bednets offered effective control, which resulted in a minimal variation of incidence in this age group. The calculated prevented fraction by bednets over DDT is 76.23%

The mean monthly incidence in the 5-9 year olds are in Fig.75. The trend with this age group started higher prior to bednets. But with bednets the mean incidence declined and was

**Fig.74:** *Mean monthly malaria incidence in the 1-4 year old, in the intervention group.*



**Fig.75:** *Mean monthly incidence in the 5-9 year old in intervention areas.*



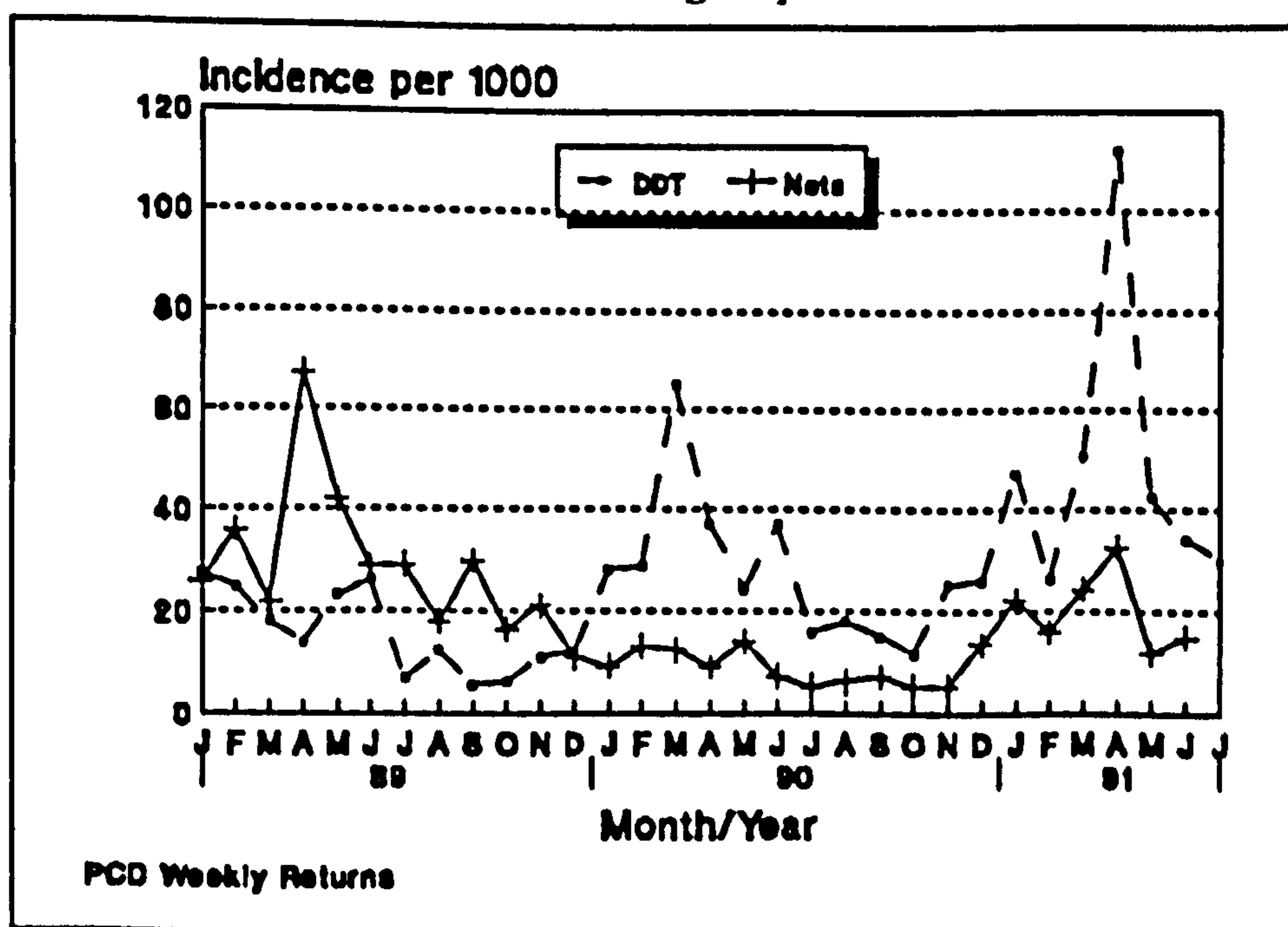
maintained at a low monthly mean of 15.4 cases per 1000 population of 5-9 year old. The rise in the first quarter of 1991 was mainly with P.vivax and in older age groups. In the DDT area,



even though the incidence was lower in 1989, it rose with peaks during high transmission season, especially in 1991. The variation indicated that DDT was not able to contain transmission as affected by other variables to a monthly mean incidence of 46.9 cases per 1000 population of the 5-9 year old. The prevented fraction of malaria by bednet over DDT in this group was 67.15%.

The trend of mean monthly incidence in the 10-14 year old are in Fig.76. The picture was again similar to that of the previous age group. The incidence was higher prior to bednet introduction, but was brought down and maintained at a low monthly mean of 20.79 per 1000 population of 10-14 year old. The incidence in the DDT area however, started lower, but

**Fig.76:** *Mean monthly malaria incidence in the 10-14 year old in intervention groups.*

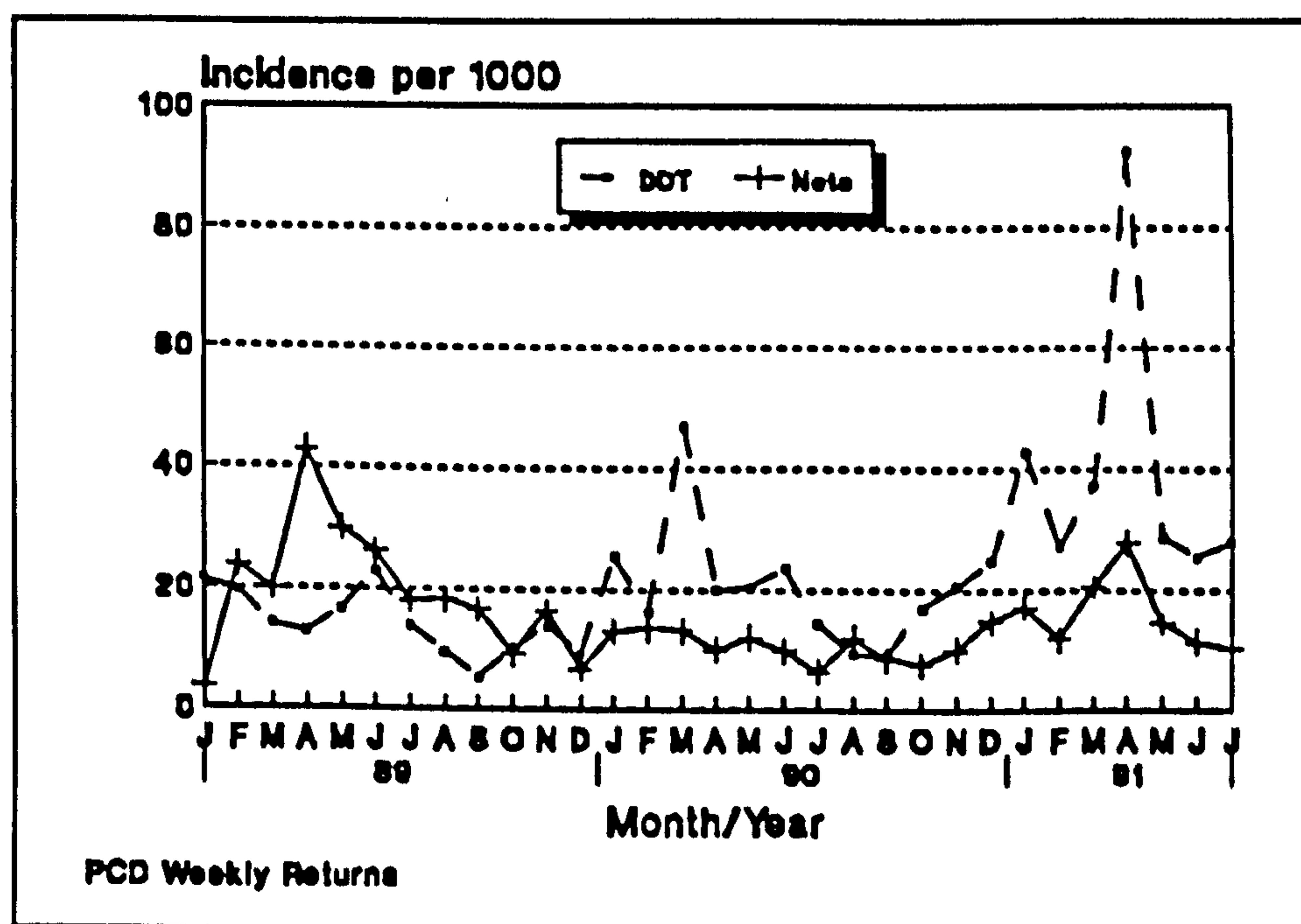


continued to rise, with peaks during transmission seasons to the highest in 1991, with a mean monthly incidence of 49.0 per 1000 10-14 year old. It was obvious that DDT was not able to contain transmission in this age group. The calculated prevented fraction of malaria by bednets over DDT in this age group was 57.57%.

The trend of monthly incidence in the 15 years and older in the intervention groups are

shown in Fig.77. The mean monthly incidence in the bednet group followed the same pattern as the above age groups. It was higher prior to bednets but was brought lower than DDT and maintained with little variation. The rise in the first quarter of 1991 was mainly with *P.vivax*, and by that time the mean monthly incidence per 1000 15 year old and older was 16.21. In the DDT area the increase started in 1990 and varied with the transmission seasons reaching a peak

**Fig.77:** *Mean monthly incidence in the 15 years and older in intervention groups*



in 1991. It was clear that DDT was not able to effectively contain the transmission in this age group, and the mean monthly incidence was 40.32 per 1000 population of 15 years and older. The calculated prevented fraction of malaria by bednets over DDT in this age group was 59.8%.

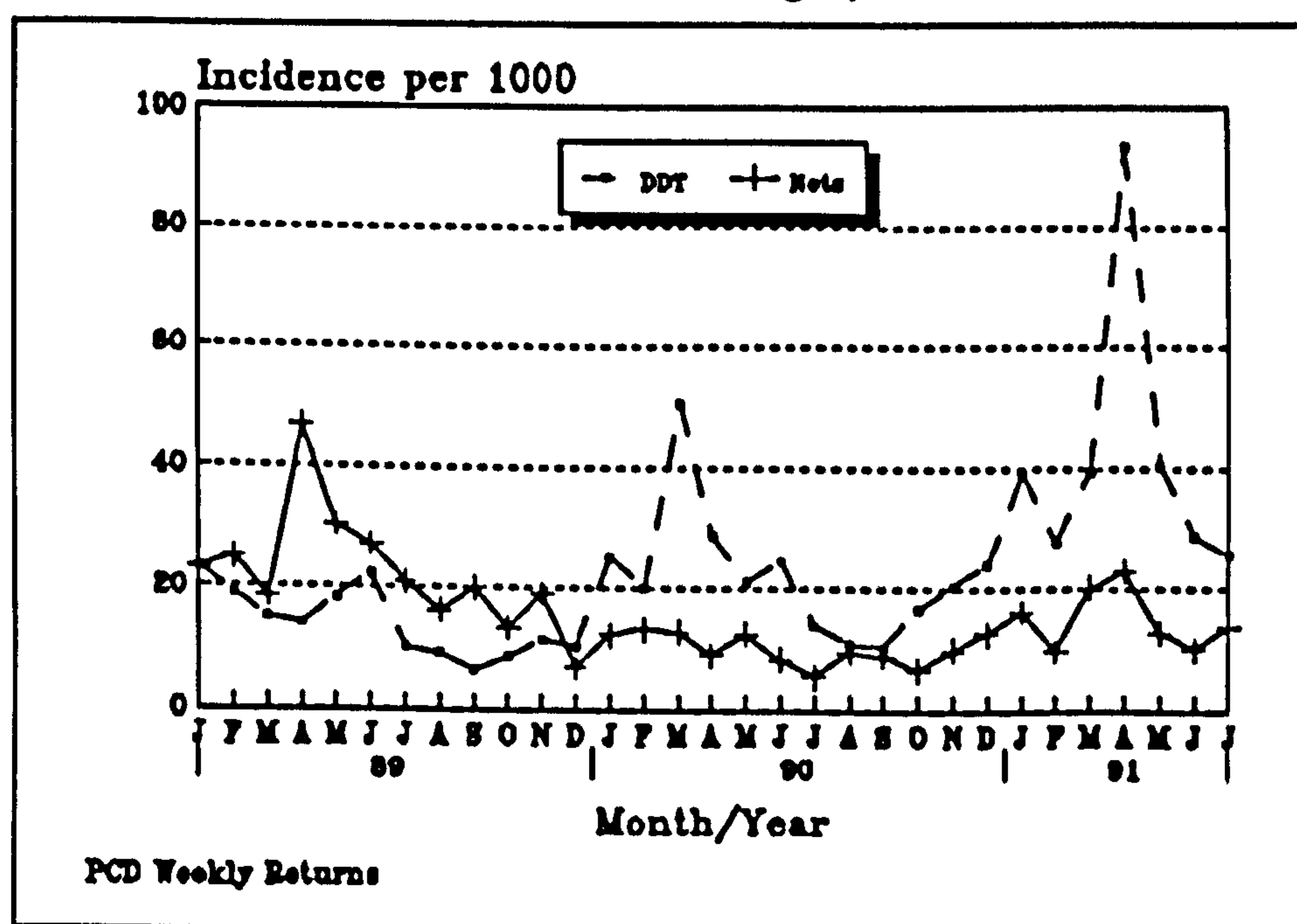
When the mean monthly incidence in all ages are combined the pattern is as presented in Fig.78. The mean incidence in the bednets, though higher prior to bednets, was brought down and maintained with little variation at a monthly mean incidence of 15.1 per 1000 population. The mean incidence in the DDT area, though lower, rose in 1990 to reach a peak in 1991. There were great variations according to the transmission season, in which DDT spraying was unable



to contain malaria as effectively it may have done in the past. The mean monthly incidence was 42.5 per 1000 population. The calculated prevented fraction of malaria in all ages by bednets over DDT was 64.5%.

Therefore from these results permethrin impregnated bednets not only, brought down malaria to a low level, but maintained it so that the incidence did not vary as much as in the DDT group. This was particularly with P.falciparum. DDT no longer provided the positive

Fig.78: *Mean incidence in all ages, in intervention areas.*



impact that it may have had in the past and was therefore less effective than permethrin impregnated bednets.

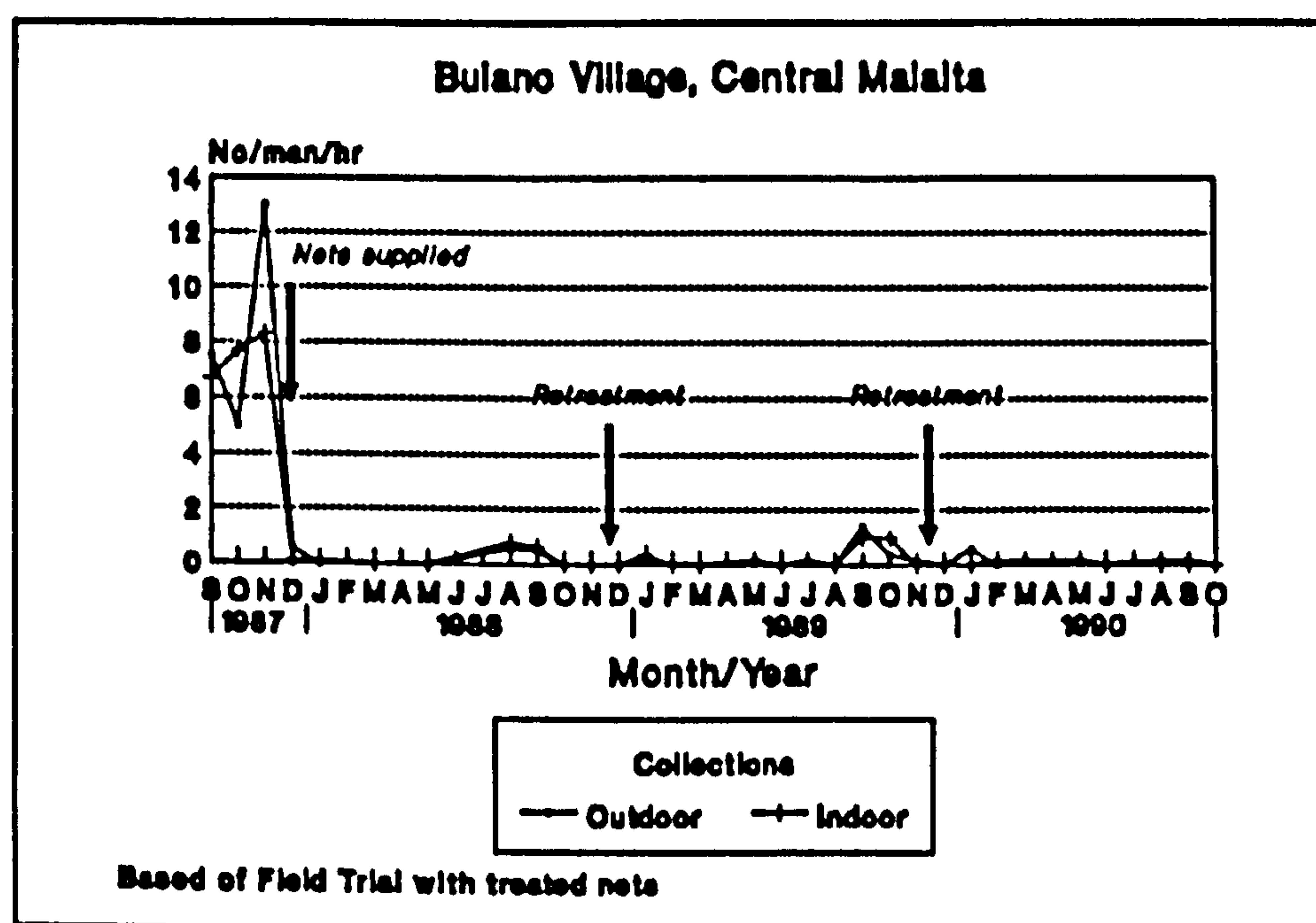
#### 4.4 THE IMPACT OF INTERVENTIONS ON THE VECTORS

A.punctulatus was still highly sensitive to interventions, especially to impregnated bednets. After pre-intervention baseline studies, (Fig.15) the density remained following impregnated bednet introduction (Fig.79). Further areas where A.punctulatus was predominant could not be studied as they were further inland and not easily accessible to regular



entomological study. Therefore almost all the entomological studies were on A.faraui, which is the most common vector transmitting malaria in the country.

**Fig.79:** A.punctulatus density in response to permethrin treated bed nets



#### 4.4.1. Changes in biting density.

These studies were done in special huts that were constructed with similar materials to village huts and were erected in each intervention area; DDT, impregnated bednets, and a comparison (no vector control interventions). Between January 1989 and July 1991 the same number of hours (126) was spent by six technicians in each area. Each time period, between 1800 hours and 2100 hours, three technicians collected all landing indoor and three technicians outdoor. The total A.faraui collected over 18 months are presented in Table 38.

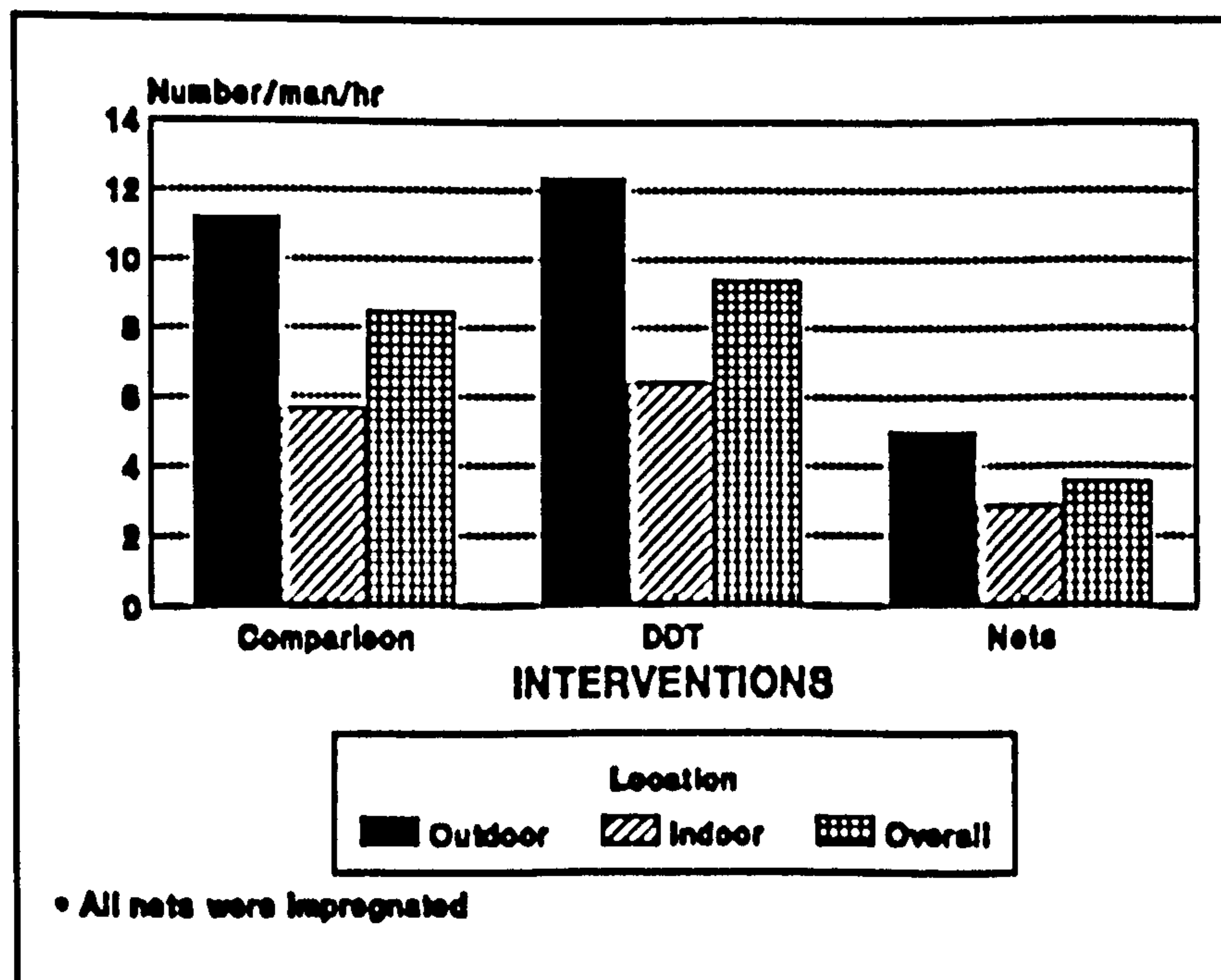
In the DDT area the mean man (collector) biting rate per hour was 12.36 outdoor, and 6.39 indoor, with an overall rate of 9.38 man biting rate per hour. In bednets area the mean man biting rate per hour was 4.97 outdoor and 2.83 indoor, and the overall man biting rate in the bednets area was 3.51 per hour.

**Table 38.** Number of A.farauti collections between 1800 - 2100 hours over 18 months, 1989-1990.

METHOD	NUMBER CAUGHT		DENSITY
Site	Outdoor	Indoor	Number/man/hour
Comparison	4224	2146	8.43
DDT	4670	2414	9.38
Bednets	1879	1071	3.51

The low density in bednets area is due to the killing effect of permethrin (Fig.80). The slightly higher proportion of vectors entering indoors to bite could be attributed to the non-irritant effect of permethrin. In the comparison area the mean man biting rates were, 11.18 per

**Fig.80:** *Mean man biting rates of A.farauti in DDT, Bed Nets and comparison areas.*



hour outdoor, 5.68 indoor, and 8.43 combined. This is a much similar results to those in the DDT area. This meant that DDT did not affect the density of the vector. It also meant that A.farauti has adopted a similar behavioral response, as with DDT, in areas no insecticides were used.



Data was also available in a special area where permethrin impregnated bednets and DDT residual spraying were used together. A total of 2561 mosquitoes were caught giving a man biting rate of 3.39 per hour. 1734 were caught outdoor and 827 indoors with a ratio of 2:1 and respective man biting rates of 4.59 and 2.19 per hour. The low number of mosquitoes caught outdoor was similar to those caught in the bednet area but the number indoor was very much lower. This could be an added effect of excito-repellency of DDT. It seemed that permethrin treated bednets reduced the density of A.farauti in a community, while DDT had no effect on density at all. The excito-repellency of DDT has already caused an adaptation of A.farauti to bite more outdoors. The general trend of biting density over the months followed a similar pattern, mostly related to rainfall. However in the area where the bednets were applied, there was a decrease in density and a lower trend continued after introduction of impregnated bednets.

#### 4.4.2. Changes in Behaviour of vectors.

The A.punctulatus population was so dramatically reduced [Fig.79] that it was not possible to continue studying any change in behaviour as was done prior to the introduction of bednets [Fig.15]. The overnight biting pattern of A.farauti did not change with introduction of bednets. It continued to bite more outdoor than indoor even though there is a slight increase in the proportion of indoor biting in the DDT and comparison areas [Table 38].

The data collected from experimental huts, fitted with exit traps, between January 1990 to July 1990, revealed that A.farauti did not rest on walls at all [Table 39]. All mosquitoes that were caught were in exit traps, and neither were collected dead on the floor nor, resting on the walls. This was the same in DDT, bednets, and control experimental huts. The likelihood that dead mosquitoes were scavenged by ants was prevented by the following measures; covering the floor with white sheets, two hourly collections, and the stilts of the huts were applied with sticky



tapes/cream to trap any ants that were climbing up. In the DDT hut, of the total of 238 mosquitoes that were collected, 52.5% were fed, none were collected from the floor, and none resting on the walls. Only 10.1% died within 24 hours. In a control hut, of the total of 301 mosquitoes that were collected, 62.8% were fed, and 11.6% died within 24 hours.

In the permethrin treated bednet hut, of the total of 223 mosquitoes collected only 43% were fed, but 98.2% died within twenty four hours. Several were found already dead inside the exit traps during collection. In another experimental hut, an untreated bednet was used.

**Table 39.** Total A.farauti and 24 hr mortality collected from Experimental Huts.

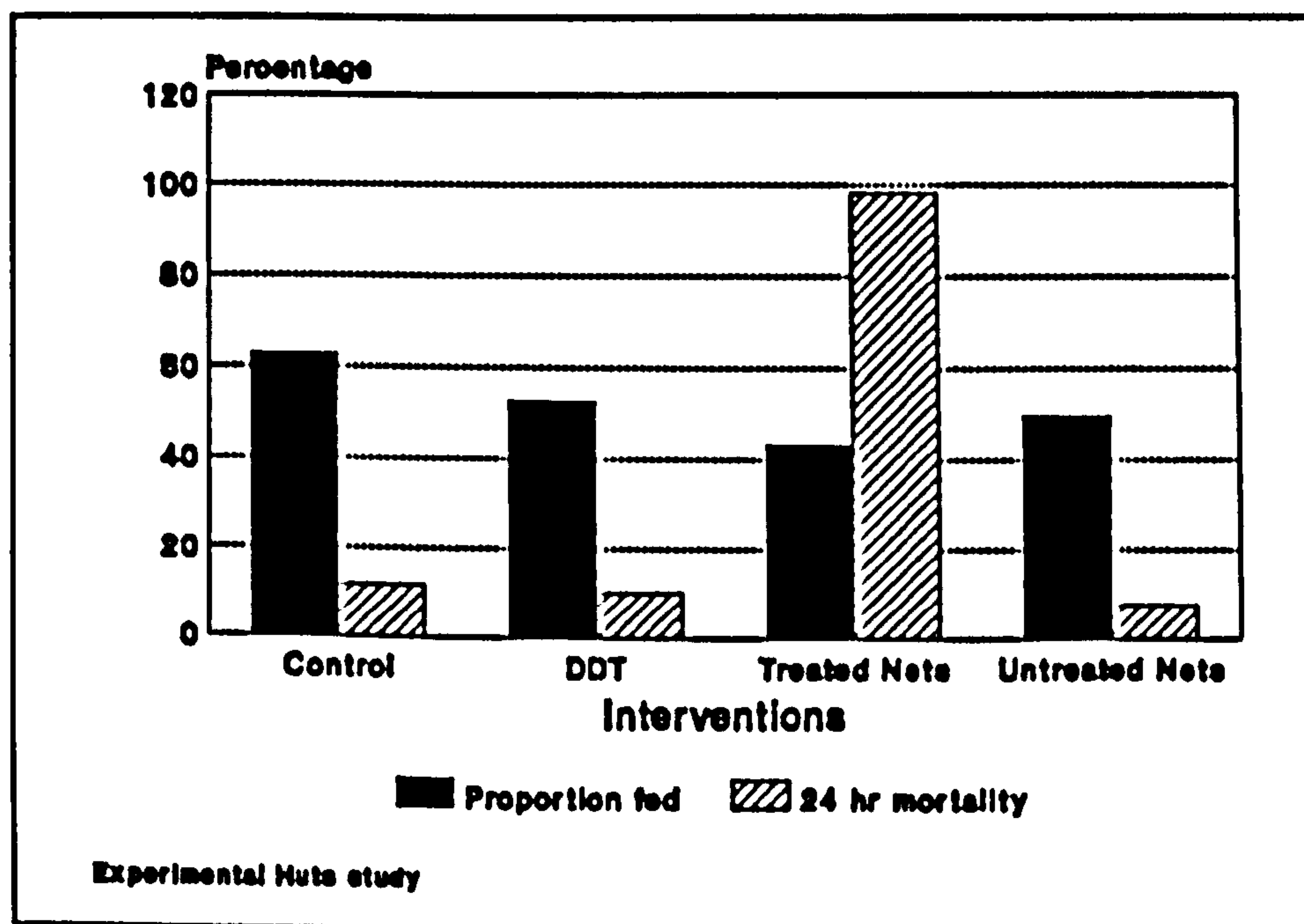
METHOD	Number collected from				24 hr mortality	
	Window traps		Walls	Floor		
	Fed	Unfed	F/U	F/U	Number	%
Comparison	189	112	0	0	35	11.6
DDT	125	113	0	0	24	10.1
Untreated Nets	116	118	0	0	18	7.7
Treated Nets	96	127	0	0	219	98.2

In this hut, with untreated bednet, a total of 234 A.farauti were collected. 49.6% were fed and none were caught resting on the walls.

Even though there is some inhibition of feeding, a higher proportion managed to feed probably through the untreated materials. Only 7.7% died within 24 hours, much lower than those that died in the comparison (Fig.81). Less mosquitoes were able to feed through impregnated bednets and almost all that came into contact died within 24 hours. This meant that permethrin impregnated bednets inhibited feeding, even though not significantly different from

untreated nets. Both the comparison and DDT results revealed high feeding rates, even though they were slightly lower in the DDT hut. Some mosquitoes may have been repelled before feeding.

**Fig.81:** *The proportion of fed A.farauti collected and 24 hr mortality..*



The 24 hour mortality showed that impregnated bednets were more effective than DDT, untreated nets and comparison. All mosquitoes that fed tried to escape to rest outside instead of resting on the walls. It is clear that this behavioral resistance is fully established in the species, even if no insecticides were used. There was no difference in 24 hour mortality in mosquitoes collected in the DDT hut and comparison hut.

#### 4.4.3. Changes in Susceptibility of vectors

667 wild female A.farauti caught in the DDT, permethrin impregnated bednet and comparison areas, between February and May 1990, were subjected to the standard WHO bioassay tests (Table 40). The mosquitoes were exposed to the same insecticide applied on the area from which they were caught. 94 were exposed to permethrin, 413 to DDT, and 160 as



comparison. The mortality in the comparison group was 7.5%, 77.0% with DDT, and 100% with permethrin. By Abbott's correction the mortality with DDT was 75.13% and permethrin 100%.

**Table 40. Results of Bioassay Tests done with wild caught mosquitoes in intervention areas (between February to May 1990).**

AREA	Number used	Dead	Alive	% Mortality
Comparison	160	12	148	7.5
DDT	413	318	95	77.0
Bednets	94	94	0	100.0

These results show that, even though there may have been an increase in tolerance of A.faraudi to DDT, there is still a significant killing effect of the insecticide. The low 24 hour mortality of mosquitoes in the exit traps collected from the DDT hut (Table 39) further confirmed that A.faraudi has totally avoided contact with DDT sprayed surfaces. It exited immediately after feeding to rest outside, thus displaying a well adapted exophilic behaviour, even though it may be highly endophagic.

#### 4.4.4. Changes in Infective potential of vectors

##### a. *Parous Rates*

The general parity was calculated by dissecting ovaries of A.faraudi mosquitoes. A total of 12,734 A.faraudi female mosquitoes were dissected, 5837 caught from DDT area, 2118 from impregnated bednets area, and 4779 from comparison area (Table 41). The samples were collected from both outdoor and indoor in each area. In the DDT area the mean parous rate indoor was 54.1% and outdoor 53.0%. The respective standard deviations were 8.33 and 8.63, and standard errors 0.13 and 0.20. The 95% confidence interval of the means were, 52.74% to 53.26%, and 53.71% to 54.49%, respectively, for outdoor and indoor.



In the impregnated bednets area the mean parity indoor was 48.6% and outdoor 51.2%.

The respective standard deviations were 7.87 for outdoor and

**Table 41. Mean Parity of *A.farauti* caught between March 1989 to December 1990, and their standard deviation, standard error, and 95% confidence interval.**

	Site of Collection	DDT	Bednets	Comparison
Number Dissected	Outdoor	3926	1276	3154
	Indoor	1911	842	1625
Mean parity	Outdoor	53.0	51.2	53.4
	Indoor	54.1	48.6	55.0
Standard deviation	Outdoor	8.33	7.87	8.56
	Indoor	8.63	7.40	8.92
Standard error	Outdoor	0.13	0.22	0.15
	Indoor	0.20	0.25	0.22
95% confidence Interval of Mean	Outdoor	52.74-53.26	50.77-51.63	53.11-53.69
	Indoor	53.71-54.49	48.11-49.09	54.61-55.39

7.40 for indoor. The standard errors were 0.22 for outdoor and 0.25 for indoor. This gave the 95% confidence interval of means of between 50.77% to 51.63% outdoor, and 48.11% to 49.09% indoor in the area. In the comparison area the mean parous rate outdoor was 53.4% with standard deviation of 8.56 and standard error 0.15. In the indoor samples the mean parity was 55.0% with standard deviation of 8.92 and standard error of 0.22. The respective 95% confidence interval of the mean were 53.11% to 53.69% and 54.61% to 55.39% for outdoor and indoor.

This analysis showed no significant difference between comparison area and DDT. The indoor parous rate in both areas even though not significant tended to be higher than respective outdoor rates. This would indicate a slightly higher risk of being inoculated indoor than outdoor.

A high proportion of the mosquitoes that contributed to high biting density outdoors (Table 38) were nulliparous, which subsequently gave rise to lower outdoor parity. Even though the mean parous rates are not the same, the 95% confidence intervals overlap confirming that there is no differences in general parity of the vector populations in these two areas. This would indicate the risk to be infected in the DDT area was not different from the area without intervention.

In the impregnated bednets area, even though there was already a lower density, the parous rates outdoor and indoor were both lower than those in the comparison and DDT areas. The indoor parity is especially low. This indicated that a higher proportion of vectors in bednets areas, especially those that entered to bite, were nulliparous. It would seem that, permethrin, in killing mosquitoes, reduced the parous rates. This would affect vector survival by also reducing longevity of vector populations. Therefore the risk of getting infected was reduced in impregnated bednets areas, in particular, indoor. In comparing the differences between means of impregnated bednets and DDT there is a significant difference between the two ( $p < 0.05$ , Chi Sq.) and the respective 95% confidence intervals do not overlap, confirming the 95% chance that the vector population were different in response to interventions. The chance of being infected by the current vector population was higher in the DDT areas than in impregnated bednets areas.

#### b. *Sporozoite Rates*

The sporozoite rates of 2045 samples (thoraces) of A. farauti collected between 1 January 1988 and 1 May 1989 by ELISA (Table 42) were as follows. In the DDT area, of the total of 840 samples that were tested, 559 were from outdoor and 281 indoor. The sporozoite rate outdoor was 0.18%, all P. vivax, and indoor was 1.42% (P. falciparum 3, P. vivax 1). The average sporozoite rate in the DDT area was 0.6%. For the impregnated bednets area 40 samples were tested, 11 outdoor and 29 indoor. None demonstrated any sporozoites. Even though the results



were useful, the samples were small.

Table 42. Sporozoite rates (ELISA), of mosquitoes, (A.faraui) collected between 1 January 1988 to 1 May 1989.

Sites		Out	In	Total
DDT	Number	559	281	840
	Positive	Pv=1	Pv=1 Pf=3	5
	S.R. %	0.18	1.42	0.60
Bednets	Number	11	29	40
	Positive	0	0	0
	S.R. %	-	-	-
Comparison	Number	764	401	1165
	Positive	Pv=3 Pf=2	Pv=2 Pf=1	8
	S.R. %	0.65	0.75	0.69
Total	Number	1334	711	2045
	Positive	6	7	13
	S.R. %	0.45	0.99	0.64

In the comparison area 1165 samples were tested, 764 were from outdoor and 401 from indoor. The sporozoite rate outdoor was 0.65% (P.vivax 3, P.falciparum 2), and indoor was 0.75% (P.falciparum 3, P.vivax 1). The average sporozoite rate in the area was 0.69%. Even though the average rates, were similar in the control and DDT areas, the rate indoor in the DDT area was almost twice that of indoor in the comparison area. But the outdoor rate in the comparison area was three times less than the outdoor rate in the DDT area. This meant that the majority of mosquitoes repelled by DDT would be nulliparous and young adults. The more mature vectors bite indoor contributing to higher parity indoor in DDT and comparison areas (Table 41), as well as their respective higher sporozoite rates.

The species specific rate would indicate that transmission of P.vivax may have taken



place more outdoor, whilst transmission of P.falciparum may have taken place more indoor, particularly when DDT spraying was applied. As would be expected the most infective mosquitoes biting outdoor, repelled by DDT, were young adults with infective P.vivax parasites. P.vivax needed a shorter vectorial longevity to complete its sporogonic cycle to become infective. The more mature mosquitoes remained to bite indoor in the DDT area, and this contributed to higher sporozoite rates, and transmission of a possible higher proportion of P.falciparum which needs a longer sporogonic cycle to be infective.

Of the total 1334 samples that were caught outdoor in all areas, 6 were infected (P.vivax 4, P.falciparum 2) with a sporozoite rate of 0.45%. The total indoor sample for all areas was 711 with 7 positive sporozoites, (P.falciparum, 4 P.vivax 3) giving a sporozoite rate of 0.99%. This indicated that, in general, a higher transmission took place indoor, in particular with P.falciparum. The average sporozoite rate of 0.64% of total samples reflected a moderately high level of malaria transmission in Solomon Islands. For every 1000 A.farauti females that bite, theoretically there is a chance that 64 will produce inoculations. This is increased to 99 if biting took place indoor, and 45 if biting took place outdoor. The majority of transmission that took place outdoor in DDT sprayed area would be P.vivax which would also be higher indoor, without any intervention.

#### 4.4.5 Changes in residual permethrin concentration over time.

The residual concentration of permethrin and mortality data on impregnated netting materials are presented in Table 43. The concentration after initial impregnation were slightly higher than the target dosage of 0.5g/m<sup>2</sup> up to two months, for both samples from top and bottom sides of the bednet. Mortality remained 100%. But by the 5th month the concentration on the bottom and sides of the nets had dropped to 0.12g/m<sup>2</sup> while on the top, it was still

0.5g/m<sup>2</sup>. The mortality remained at 100% with samples from top, but the bottom and sides had declined by an average of 15%. The picture remained similar at the 9th month, but by the twelfth

**Table 43: Residual Concentration and efficacy of permethrin on netting materials, between 22 August 1989 to April 1990.**

Nets	Months in Use	Average dose per one square metre		Average mortality compared to control	
		Top	Bottom	Top	Bottom
Nylon *	0	0.56	0.66	100	100
Poly **	0	0.55	0.77	100	100
Nylon*	2	0.79	0.96	100	100
Nylon*	5	0.52	0.12	100	87.5
Nylon*	9	0.53	0.14	100	80.5
Nylon*	12	0.19	0.12	87.3	81.2

\* - All Thailand nylon netting materials

\*\* - Philippine polyethelene netting material

Note: Two bednets were sent each time and samples of 1 sq.cm. were cut from sites for tests at AMRU. Materials of 40 denier. *A.faraudi* from AMRU colony was used for mortality testing.

month the surface on the top dropped to less than 0.2g/m<sup>2</sup>. The mortalities however remained above 80% at twelve months confirming the potency of permethrin, even at low dosage. The bottom sides were frequently touched by the users who eroded the deposits.

## 4.5 LEVELS OF KNOWLEDGE, BELIEFS AND PRACTICE ON THE INTERVENTIONS.

### 4.5.1 General

Social (KAP) surveys in the form of structured questionnaire/observations were carried out to measure the following. The level of community knowledge on; the cause and spread of malaria, the effects of DDT spraying and permethrin impregnated bednets, and the level of



compliance to the two interventions. A survey was done prior to the study (pre-bednets distribution), and repeated 14 months later, and again 20 months later. The trend of measured variables were assessed.

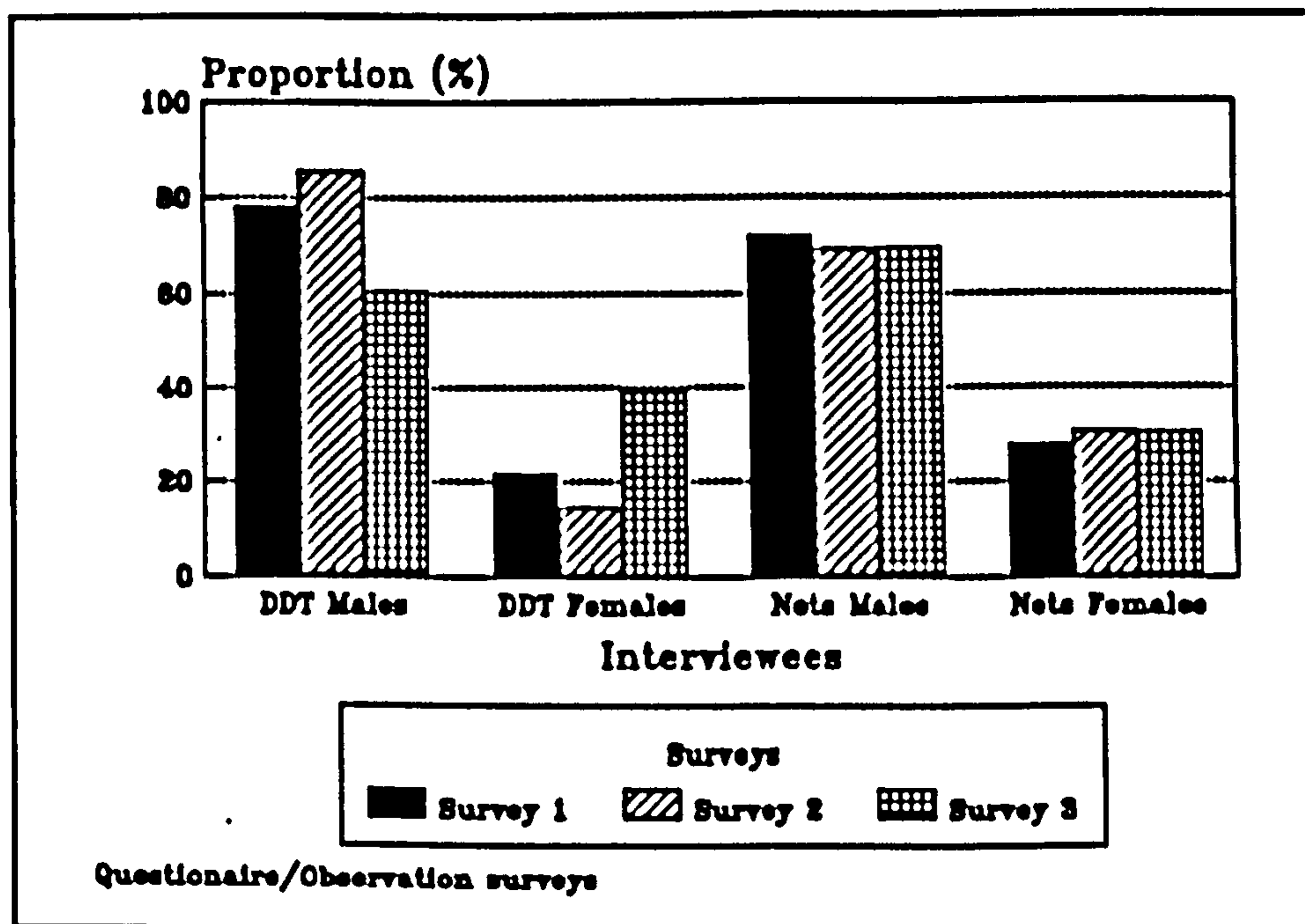
A total of 626 households in 18 villages; 9 villages in the DDT group, and 9 in the bednets group, were involved in three questionnaire/observation surveys. In the first one all villages were surveyed. In the second survey only 10 villages were randomly selected and surveyed, 5 in each intervention group, and in the last survey only four selected villages were surveyed, two in each intervention group.

The households were visited and in most cases the heads of household were interviewed. In the DDT group, 261 subjects were interviewed in the first survey (pre-bednets distribution), 159 in the second survey, and 56 in the third survey. In the permethrin treated bednets group, 365 in the first survey (pre-bednets distribution), 290 in the second survey, and 141 in the third survey.

The sex distributions of interviewees in the DDT group in all three surveys combined, were, 74.8 % males, and 25.2% females. The proportion of males increased in the second survey, but was the lowest in the last survey when more women were involved (Fig.82). In the bednets group 70.3% of interviewees were males and 29.7% females in the three surveys combined. The proportion of male and female interviewees in the bednet group remained similar in all three surveys. The average age of interviewees in all surveys combined, for the DDT group were, 27.3% below 20 years, 61.5% between 20 to 49 years, and 11.2% 50 years and older (Fig.83). The proportion of younger interviewees increased on the last two surveys with a corresponding reduction in the middle age interviewees. The older interviewees remained the same in the first two surveys but doubled in the last survey. It seemed that in this group, some middle aged people did not see any benefit of the survey, and thus left it to the younger and elderly



**Fig.82:** *Distribution of males and females interviewed in the DDT and Bed nets groups.*



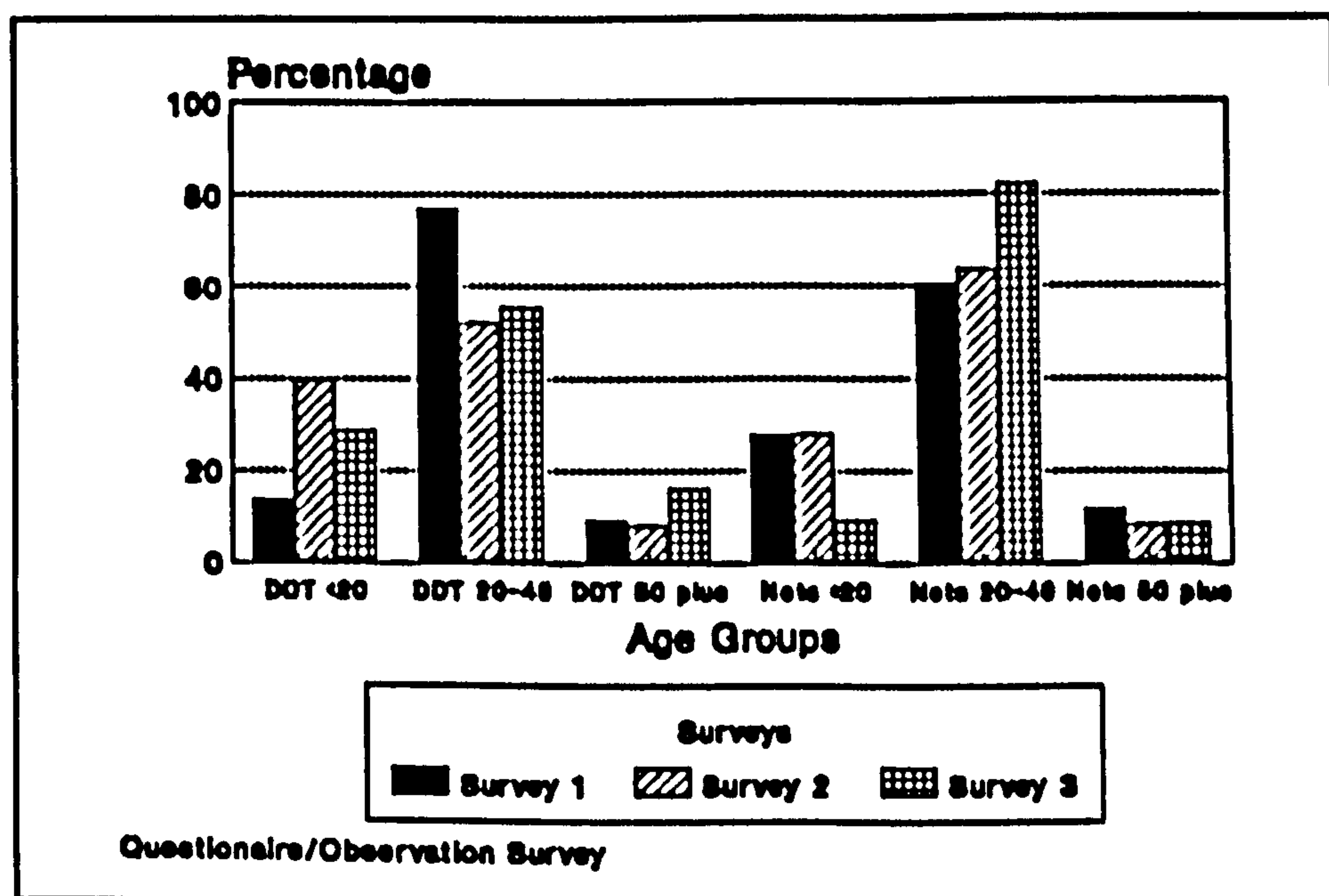
adults. Even so the majority, in all surveys, was still the middle aged adults.

In the bednets group the average age groups of interviewees in all surveys combined were, 21.6% below 20 years, 68.9% aged between 20 to 49 years, and 9.5% 50 years and older. The proportion of young interviewees, whilst remaining the same in the first two surveys, was lowest in the last survey. Only a slight decline was encountered in the proportion of elderly adults. The proportion of interviewees, aged between 20-49 years, increased to 82.3% in the last survey. Unlike the DDT group, the middle aged adults, which were the most productive age group, saw some benefit in getting themselves involved in these surveys.

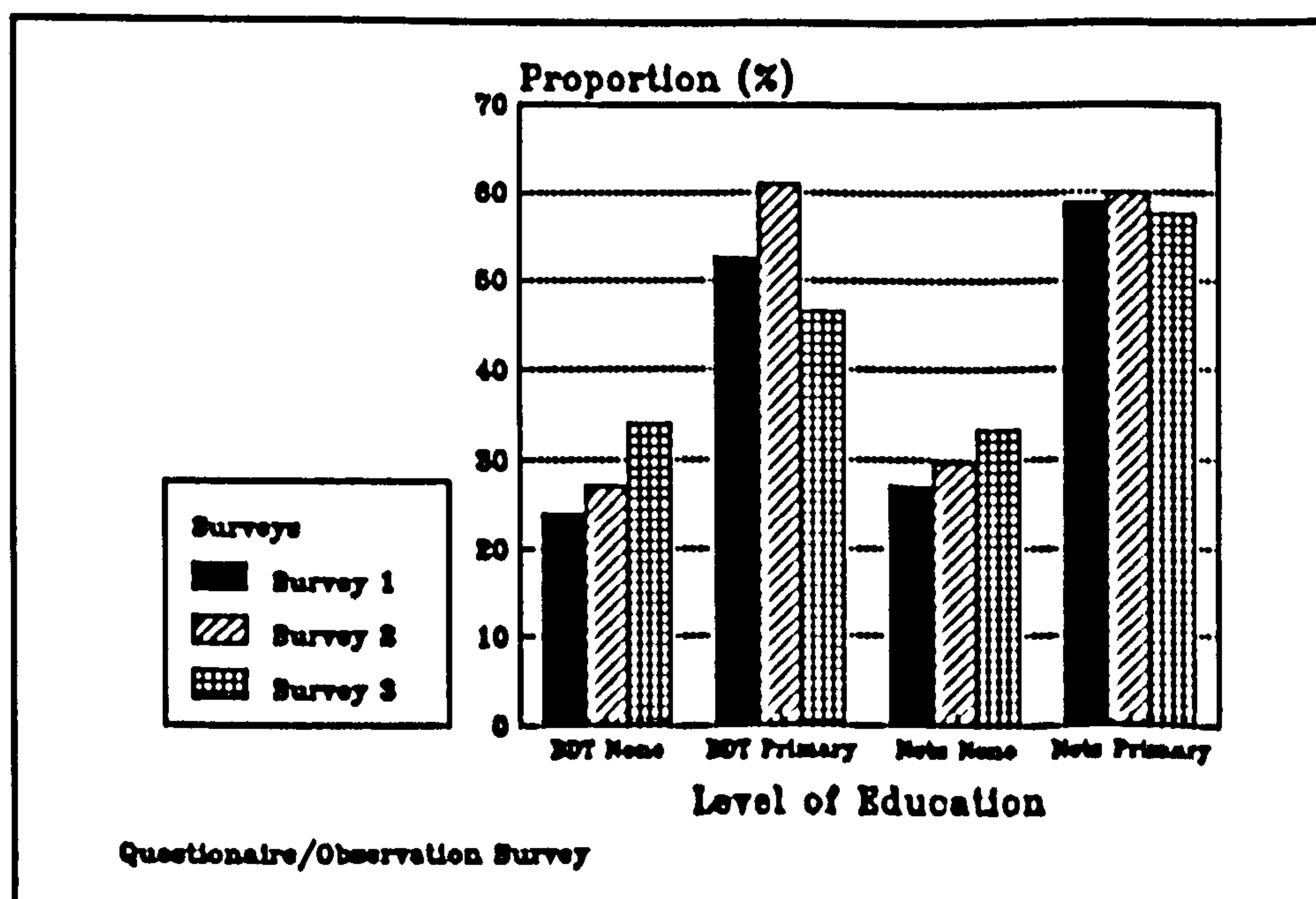
The formal education levels of interviewees in the DDT group in all surveys combined were, 53.3% primary education, 12.5% secondary education, 5.9% tertiary education, but 28.3% had none. In the bednets group, 58.8% received primary education, 7.1% secondary education, 4.1% tertiary education, and 30.0% had none (Fig.84). Those who received tertiary education in both groups mainly included school teachers, church ministers, and field agricultural workers

who mostly attended the national tertiary education (Fig.85).

**Fig.83:** *Age of interviewees in the DDT and Bed Net groups.*



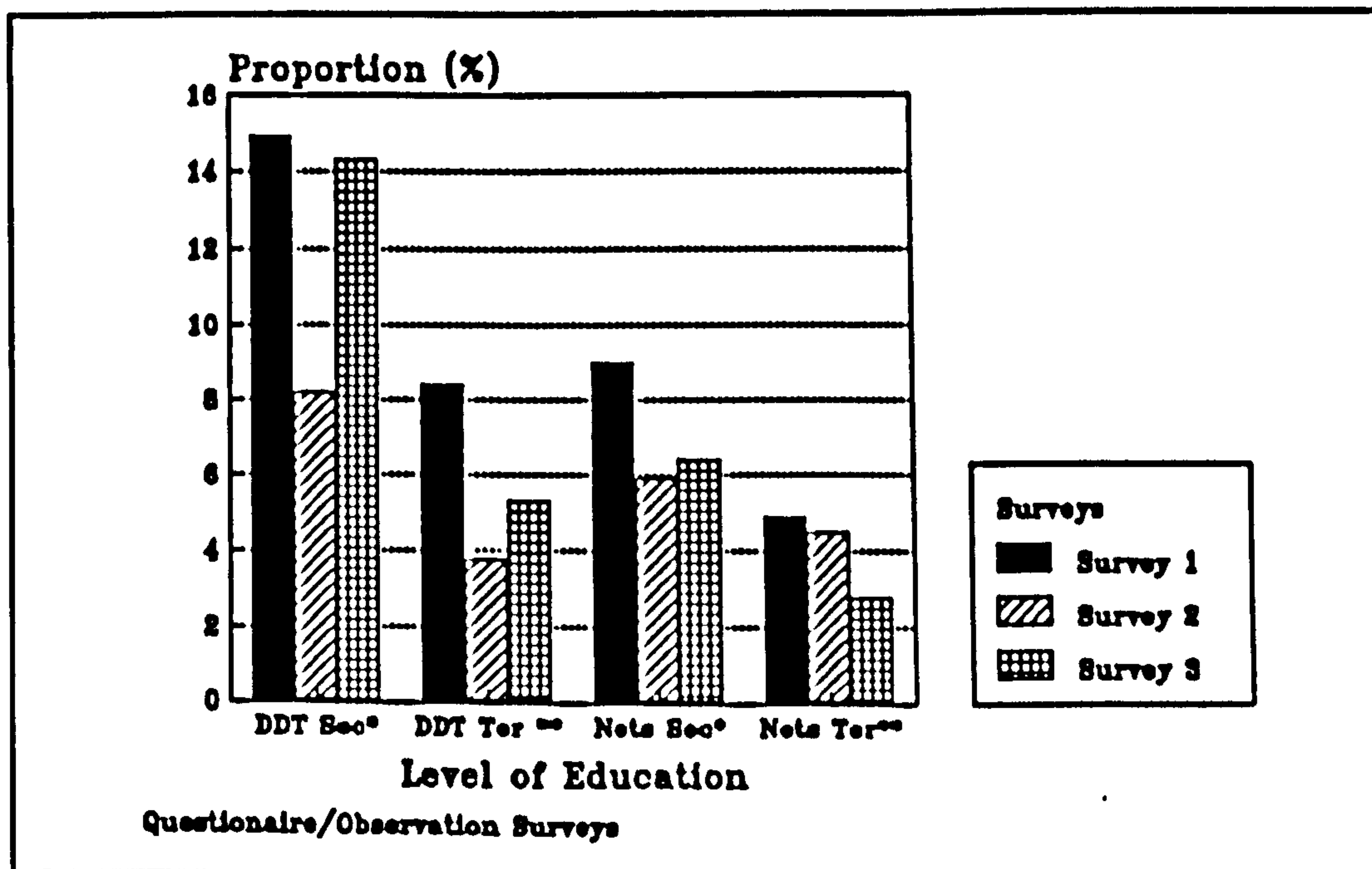
**Fig.84:** *Level of education (primary) of Interviewees.*



In both groups the main formal education was primary, and the next had no formal education (Fig.84). There was little variations in the proportion of formal education in the three surveys. Even though the proportion of interviewees with both secondary and tertiary education



**Fig.85: Level of education (secondary and tertiary) of interviewees.**



tended to be slightly higher in the DDT group, there was very little differences in the two groups (Fig.85).

#### **4.5.2 Knowledge, Beliefs, and Attitudes in DDT Group.**

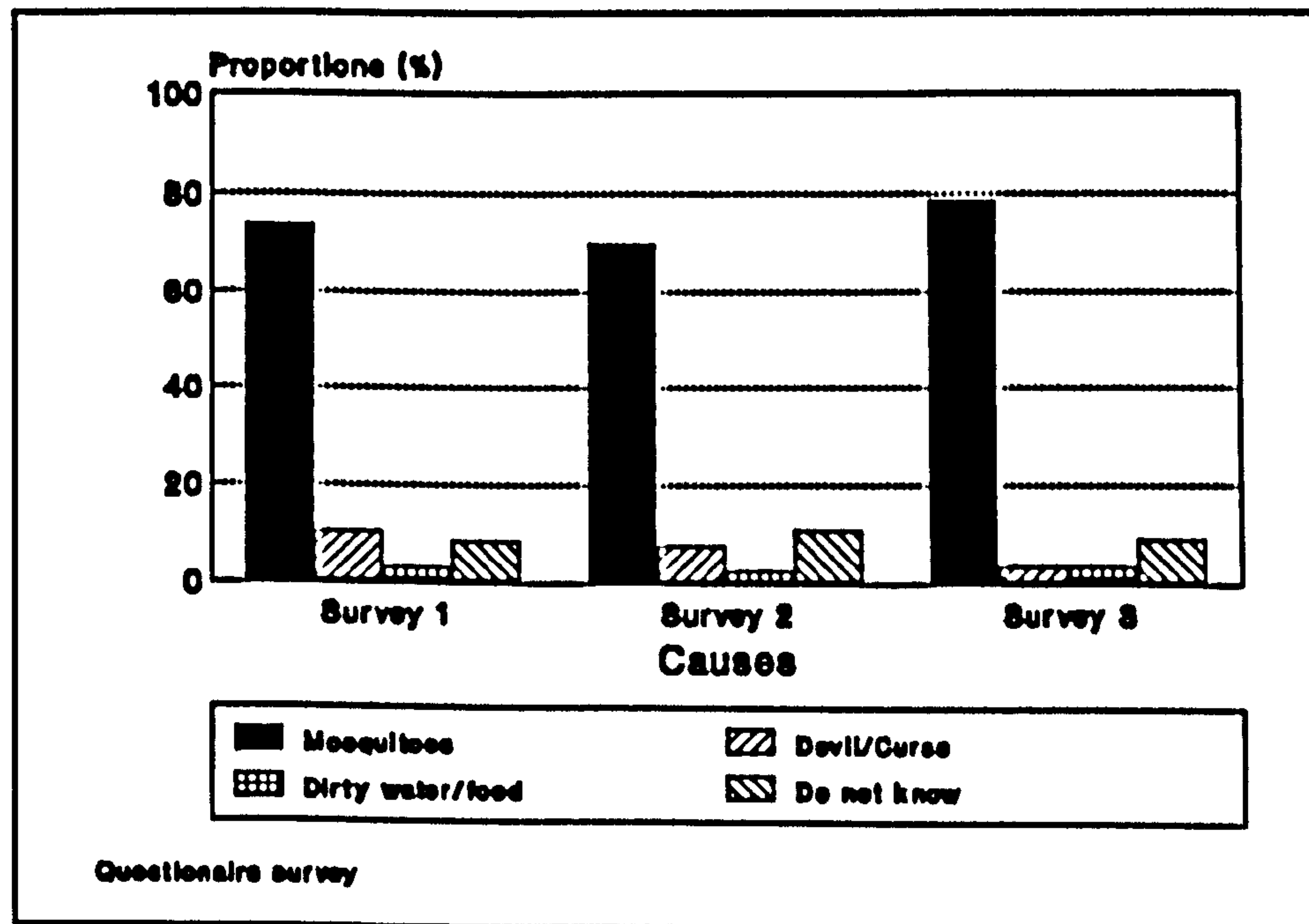
In the DDT group, an average of 74% respondents in all surveys, knew that mosquitoes spread malaria. 73.6% in the first survey, 69.8% in the second survey, and 78.6% in the third survey. The next believed the common cause was, "devil", 10.4% in the first survey, but declined to 7.6% and 3.6% in second and third surveys respectively (Fig.86). Those who did not know how malaria was spread remained about the same, 8.4% in the first survey, 10.7% in the second survey, and 8.9% in the third survey. The majority of interviewees thus knew that malaria was transmitted by mosquitoes.

An average of 72% respondents in all surveys combined allowed their houses to be sprayed with DDT during the last cycle. This was 90.8% in the first survey, but dropped to



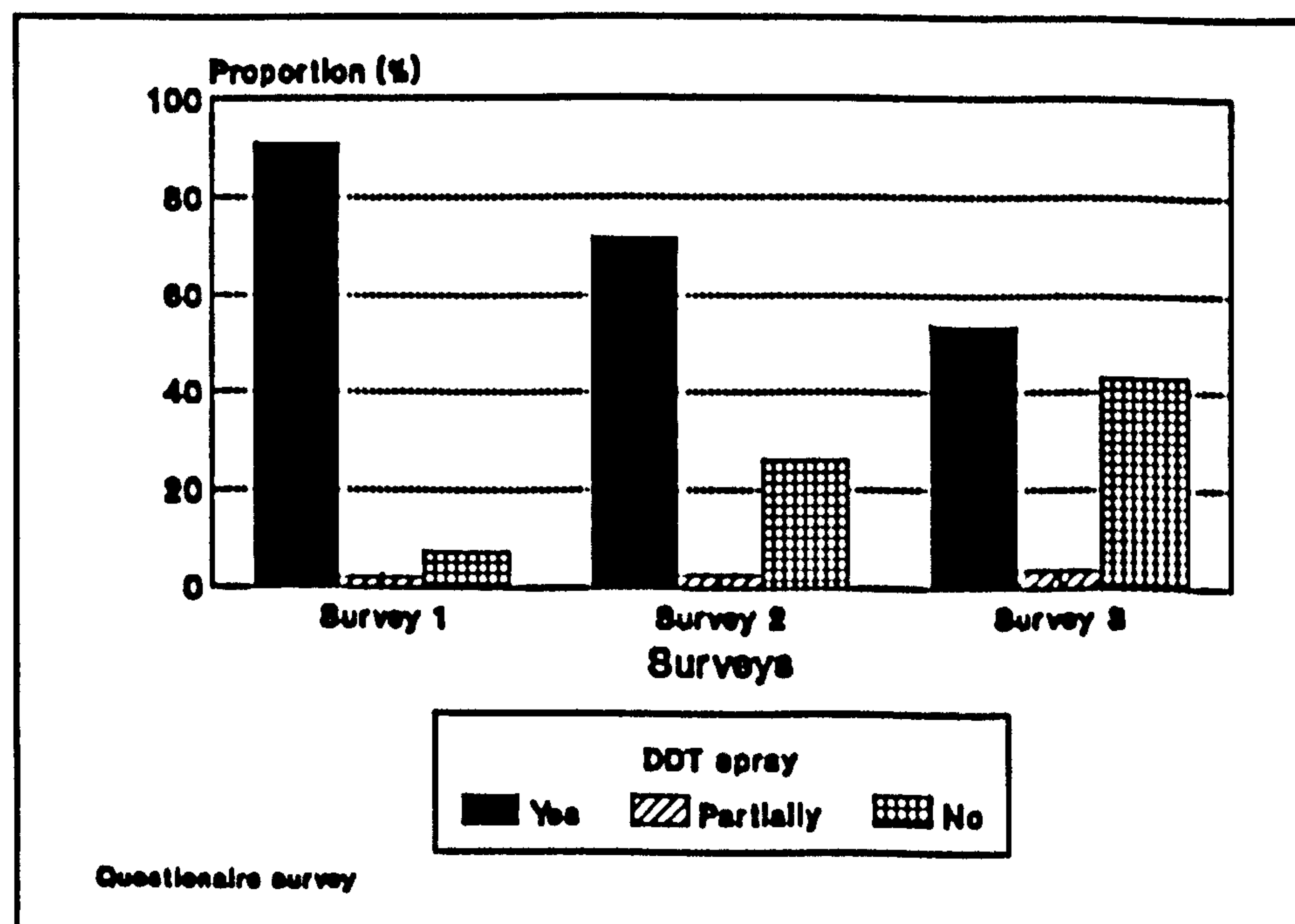
71.7% in the second survey, and 53.6% in the third survey. The increase in refusals from 7.3%

**Fig.86:** *Causes of malaria believed in the DDT group.*



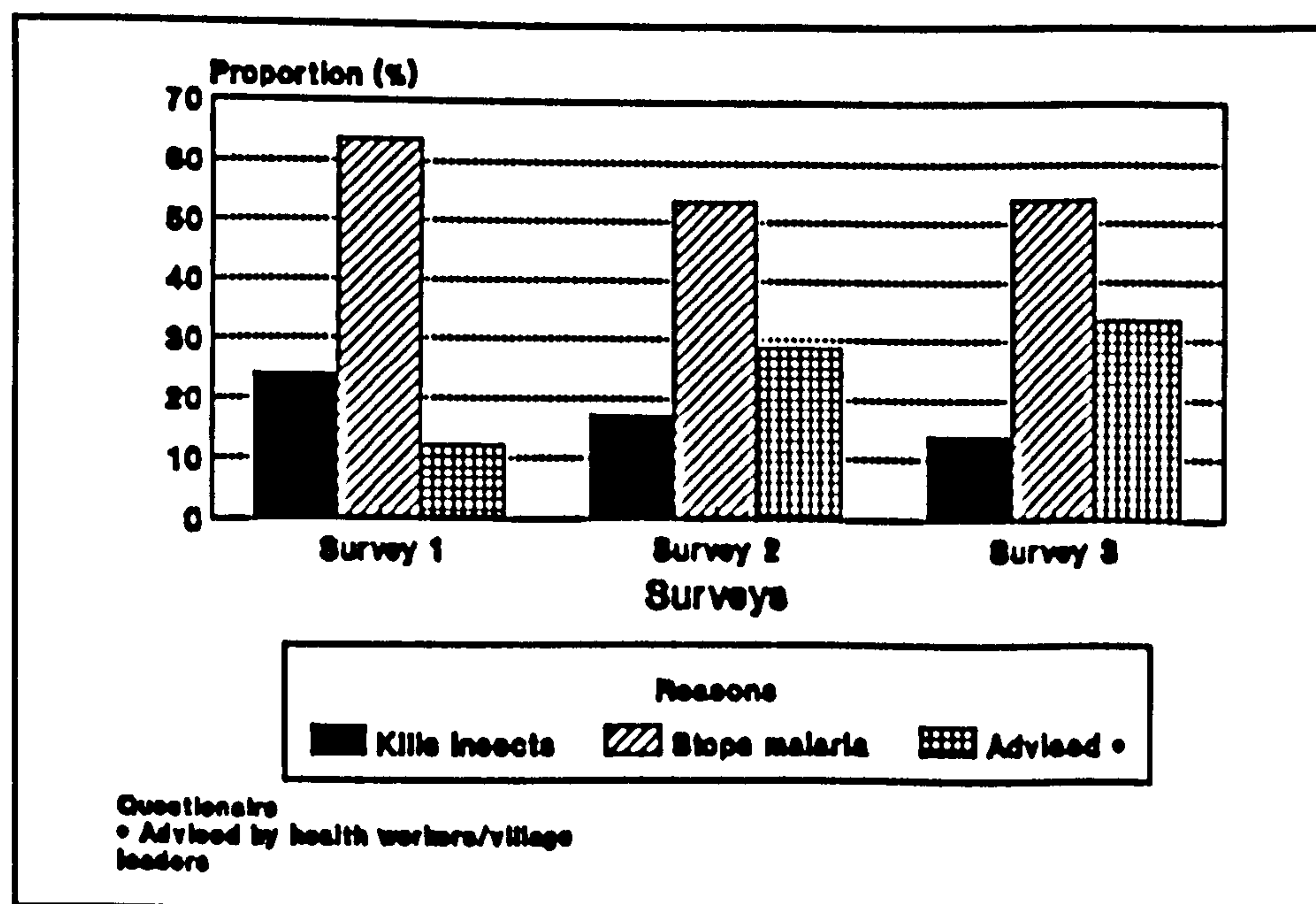
in the first survey to 42.9% in the third survey, were significant ( $p < 0.05$ ). Those who allowed only part of their houses to be sprayed were similar (Fig.87). 24% in the first survey, 17% in

**Fig.87:** *Proportion of those who allowed their houses to be sprayed with DDT.*



the second survey, and 13.9% in the third survey allowed their houses to be sprayed with DDT, because it killed all insects. Those who believed that DDT killed mosquitoes, and, therefore, prevented the spread of malaria were, 63.6% in the first survey, 53.4% and 52.8% in the last surveys respectively. But those who allowed their houses to be sprayed because they were advised to do so by health workers, and, or village leaders, increased from 12% in the first survey, to 28.8% in the second and 33.3% in the third survey (Fig.88). This suggested that as compliance to DDT spraying declined (Fig.87), those who allowed their houses to be sprayed, did so, because of advice given. Those allowed their houses to be sprayed, because they believed it stopped malaria, declined as well.

**Fig.88:** *Reasons in allowing house to be sprayed with DDT.*

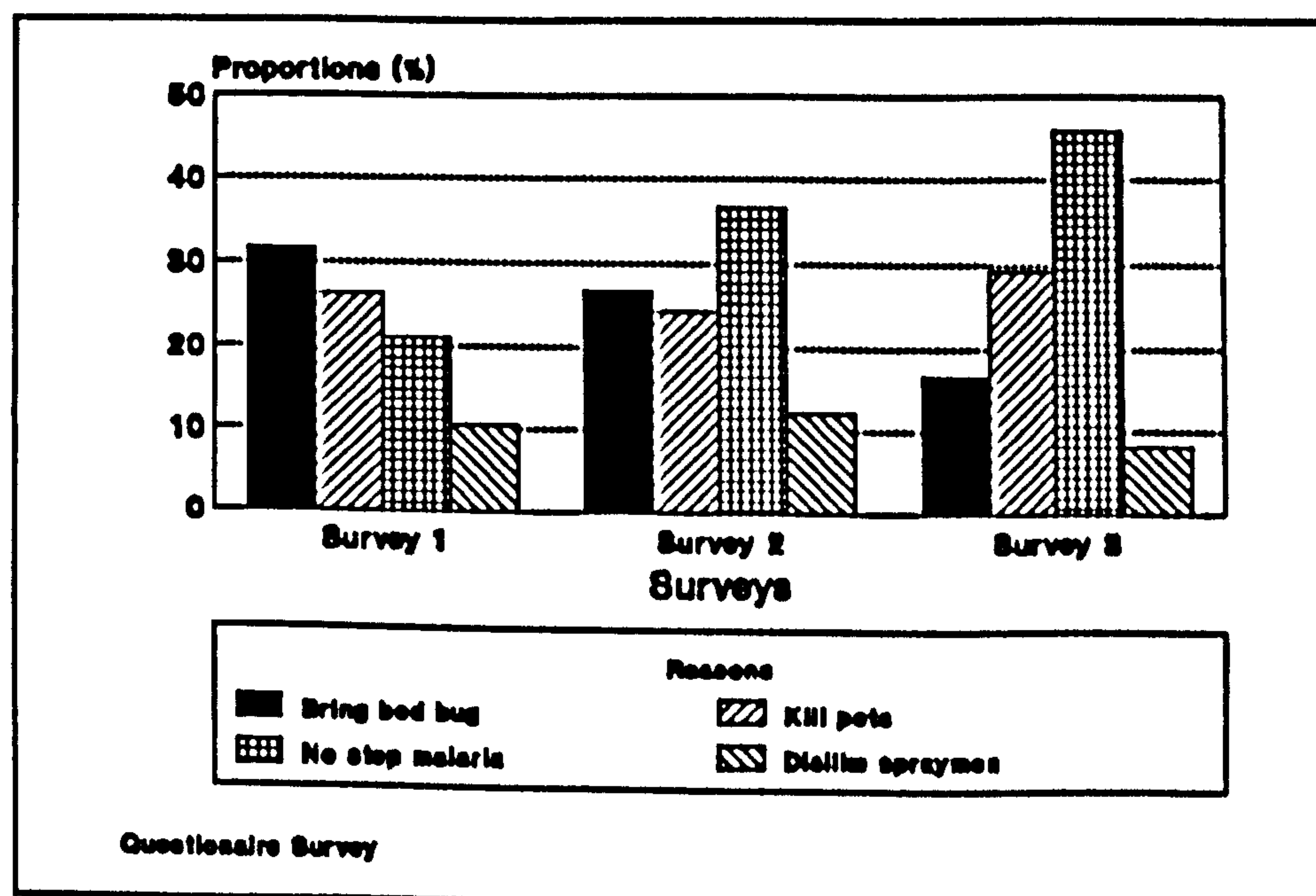


Those refusing DDT spraying gave the following reasons (Fig.89); because DDT 'brings bed bugs' 31.6% in the first survey, but declined to 26.8% and 16.7% in the second and third surveys respectively; DDT killed pets, 26.3% in the first survey, 24.4% in the second and 29.2% in the third surveys. But those who refused because they believed DDT neither killed mosquitoes nor stopped malaria illness increased from 21.1% in the first survey, to 36.6% in the second



survey and 45.8% in the third survey. This directly corresponds to the increase in refusals. There was little variation in those who refused because they disliked spraymen. The steady increase in those who refused because DDT did not 'stop malaria' which interviewees did not

**Fig.89:** *Reasons for not allowing houses to be sprayed with DDT.*



hesitate to state, was quite significant, as if they had some direct comparison with something much better than DDT.

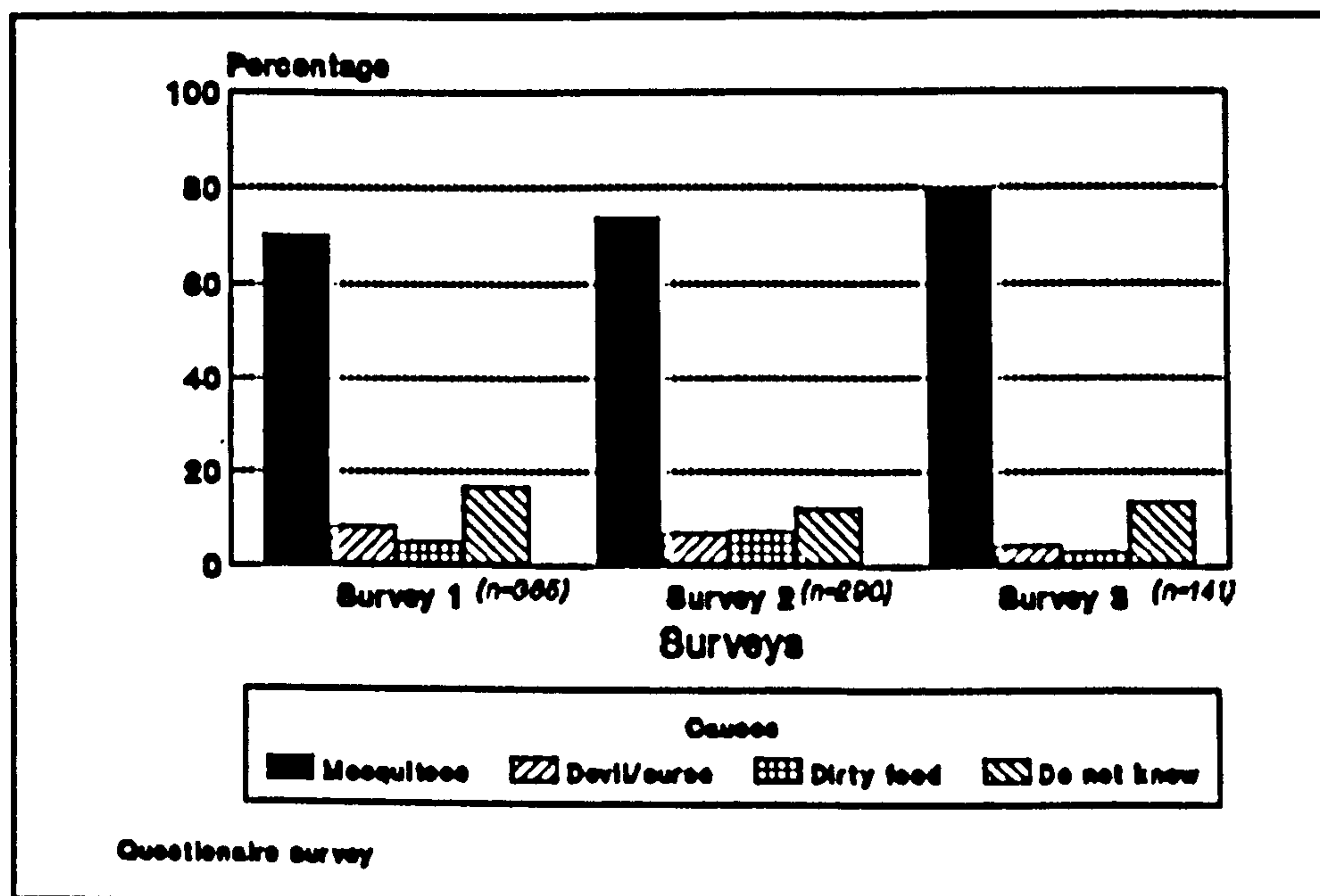
#### 4.5.3 Knowledge, Beliefs, and Attitudes in Bednet Group

In the bednets group the proportion that believed malaria was spread by mosquitoes were high, an average of above 70% in all surveys combined; 70.1% in the first survey, 73.8% in the second survey, and 79.4% in the third survey. Next common was those who did not know. This ranged from 16.7% in the first survey, to 12.1% in the second survey, and 13.5% in the third survey (Fig.90). Therefore the majority of interviewees knew that malaria was transmitted by mosquitoes.

During the first survey only 10.1% of household members claimed to have owned



**Fig.90: Main causes of malaria believed in bed net group.**



bednets. This, as expected increased to 92.8% in the second survey, and 91.5% in the third survey, after bednets were distributed. Even though 89.9% did not use bednets in the first survey, there were still some who still did not use bednets, 7.2% and 8.5% in the second and third surveys respectively. They were either missed out during bednet distribution, or did not use bednets for other reasons (Fig.92).

In the first survey, of those that used bednets, 50% used them because the nets protected them, especially babies, against insects, mainly mosquitoes (Fig.91). This declined to 21.9% in the second survey, and 22.5% in the third survey. Only 40% in the first survey claimed that they used bednets because it protected them against malaria. But this belief increased to 58.7% in the second survey, and 62.8% in the third survey. The other reasons given, that bednets kept them, especially babies, warm, and that nets provided privacy, were insignificant in the three surveys. In the first survey 79% did not use bednets mainly because they did not have them (Fig.92). This remained the commonest reason for not using bednets, 81% in the second survey, and 66.7% in the third survey. But the numbers in the last two surveys were much smaller. Another

common reason was that the nets were torn, and this remained similar in all surveys. There were

Fig.91: *Reasons for using bed nets, in the Bed Nets group.*

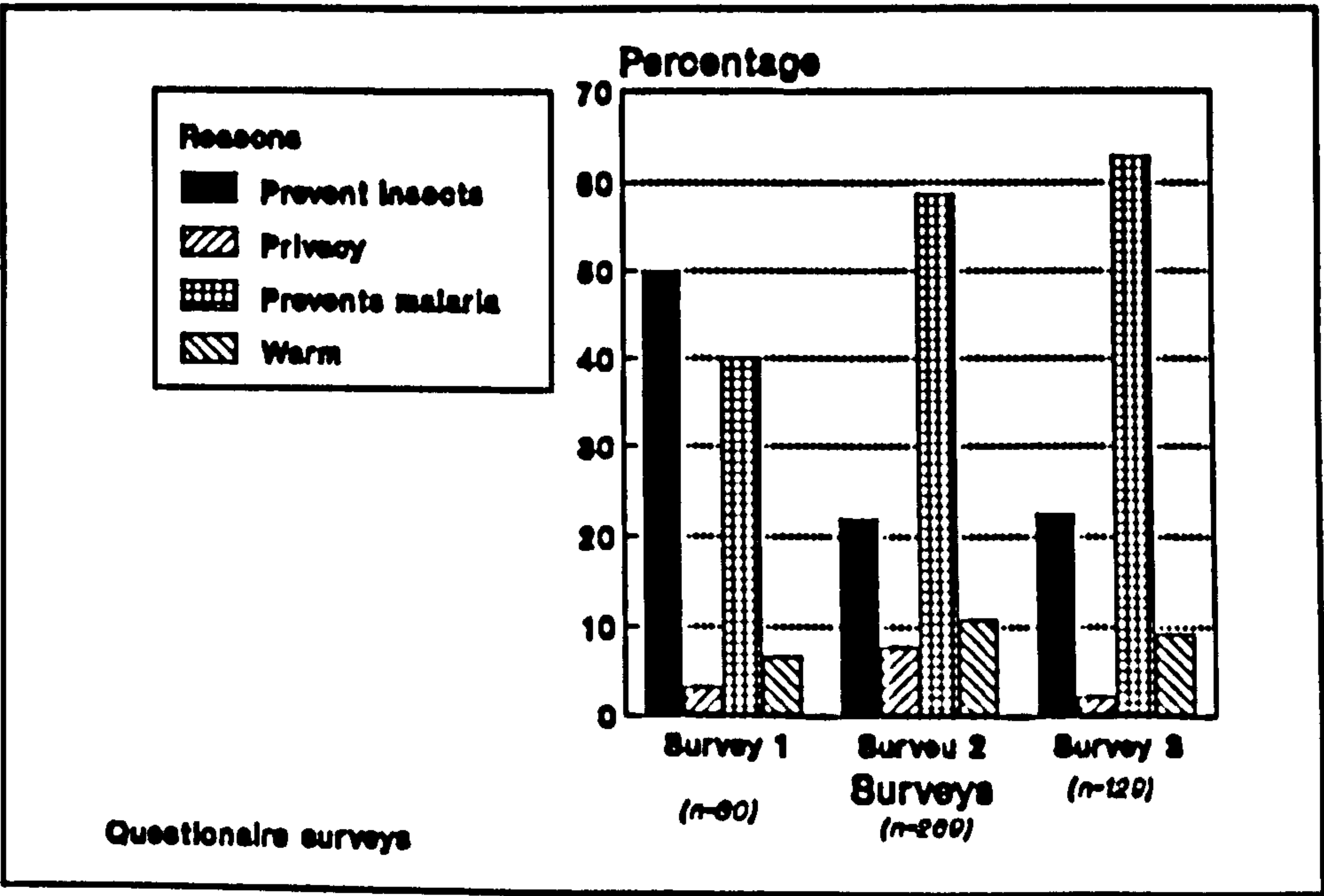
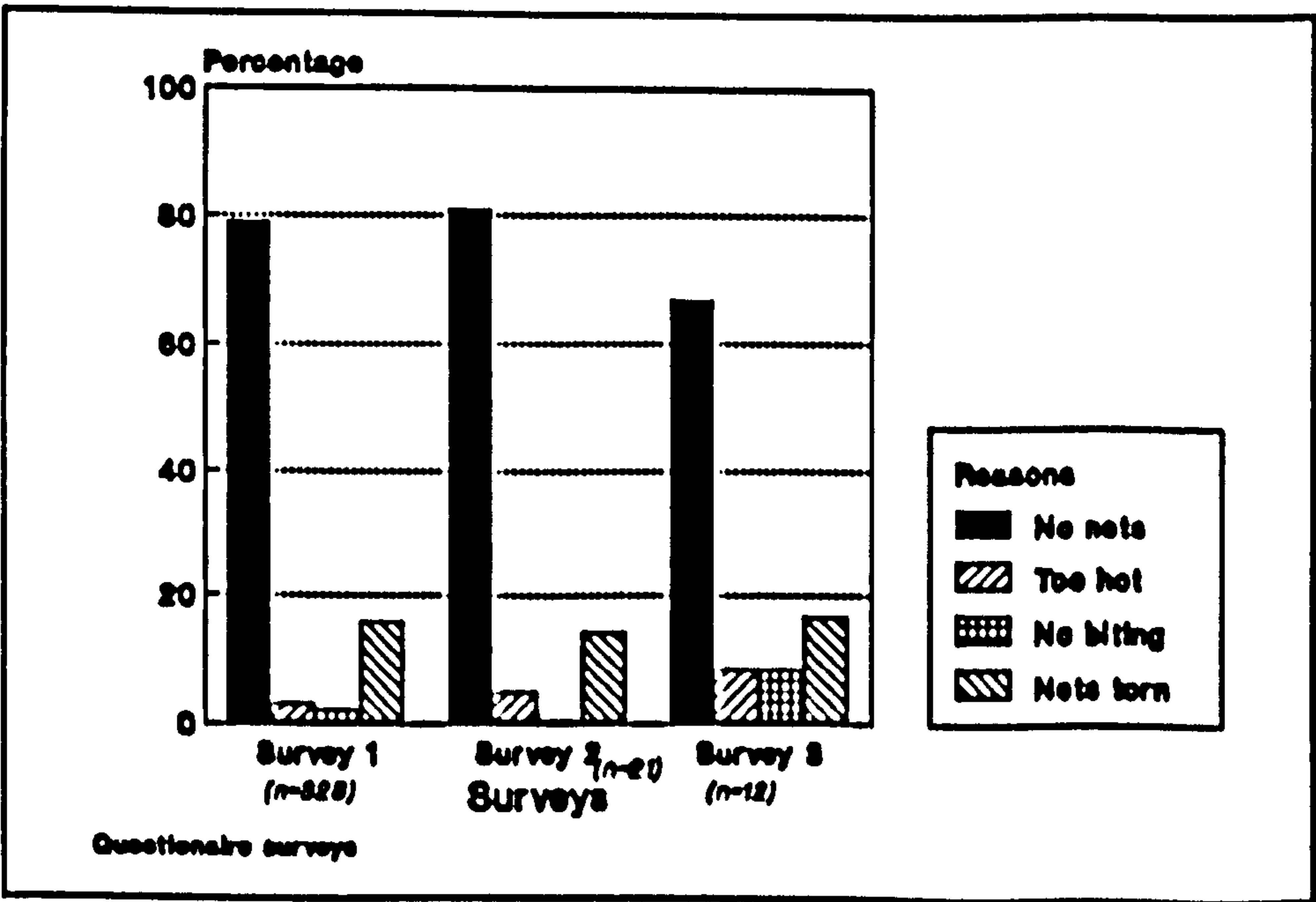


Fig.92: *Reasons for not using bed nets in the Bed Nets group.*

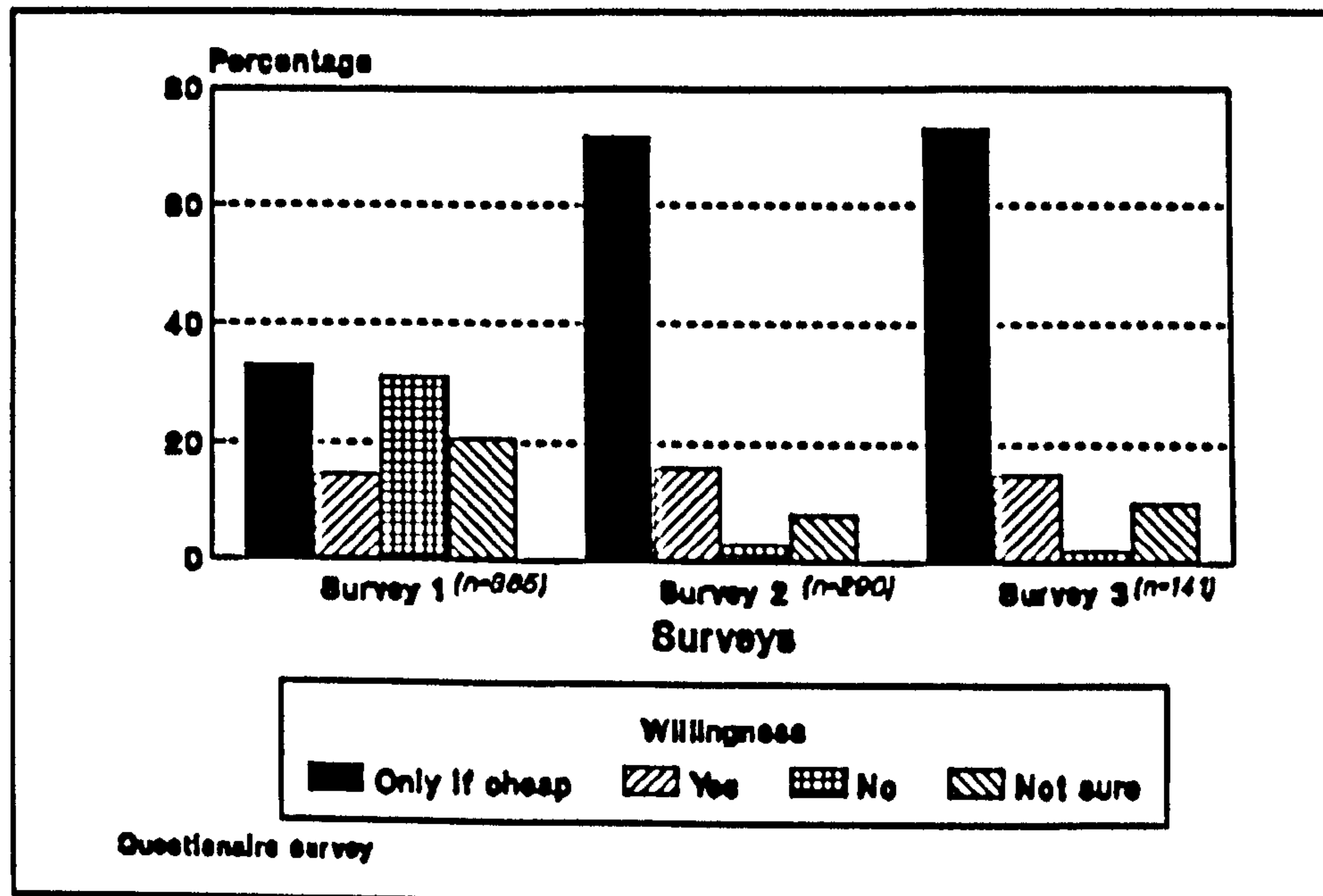


a slight increases in 'too hot' and 'mosquito biting was not a problem', as reasons in the last survey, but these were insignificant as the numbers were small. In the first survey about 33.3% of interviewees were willing to purchase bednets if they were either cheap or subsidised (Fig.93).



This increased to 71.7% in the second survey and 73.1% in the third survey. This upsurge in interest was due to observed benefit with treated bednets. Conversely those who were not willing to purchase bednets dropped from 31% in the first survey to 3.1% in the second survey, and

**Fig.93: Willingness to buy bed nets in the bed nets group.**

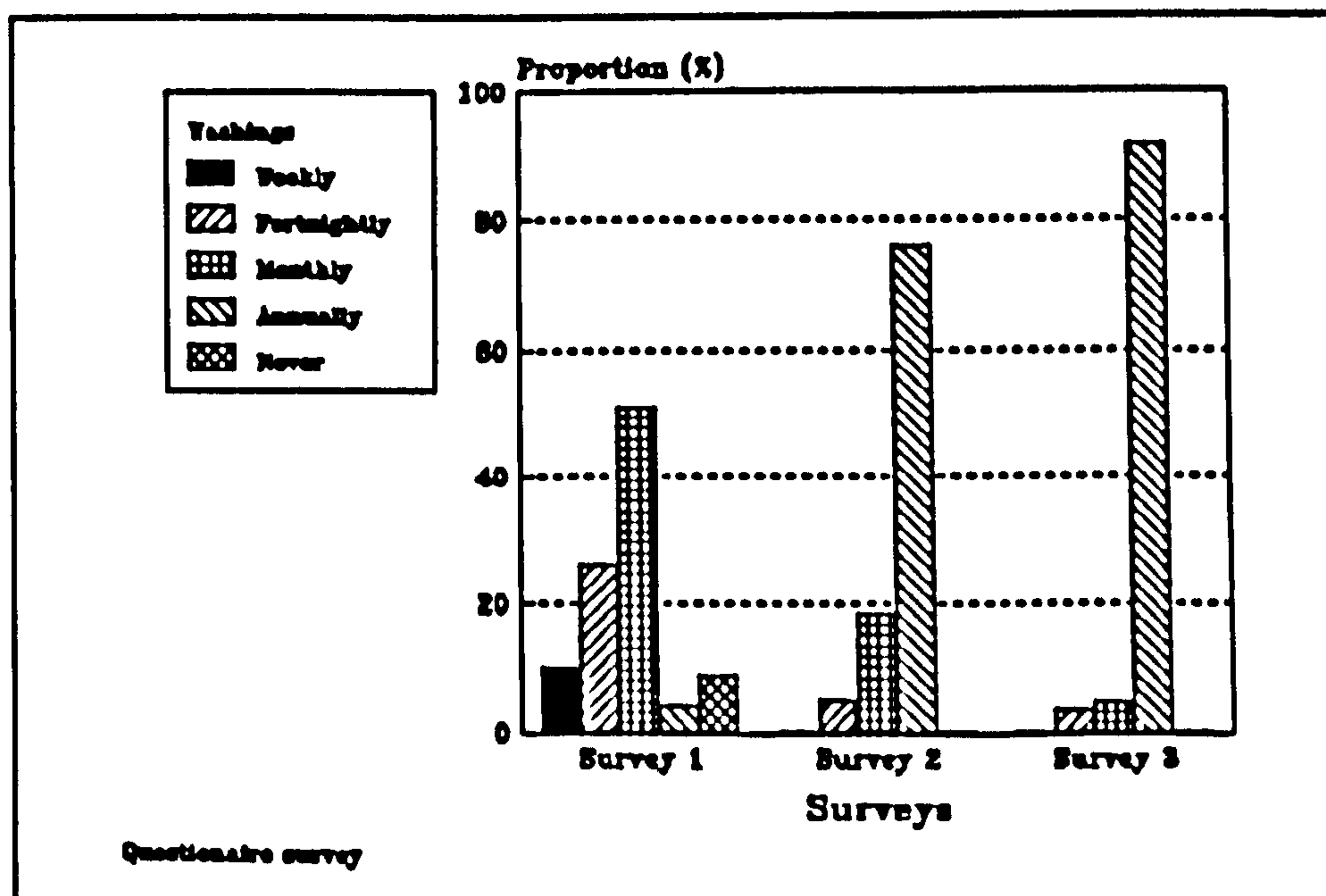


2.1% in the third survey. The willingness to buy bednets, if they were cheaper, or subsidised, was quite significant. Those that were willing to pay for bednets anytime irrespective of cost remained about the same, 14.8% in the first survey, 15.9% in the second and 14.9% in the third surveys. Those who were not sure dropped from 20.8% in the first survey to 7.9% and 9.9% in the last two surveys. Thus the majority of interviewees would be willing to purchase bednets particularly if they were subsidised.

In the first survey the majority, 51%, would wash their nets once a month, 26% once a fortnight, and 10% weekly (Fig.94). But the monthly washing declined to 18.6% of interviewees in the second survey, and 5% in the third survey. A mere 4.1% washed their bednets annually in the first survey, but this increased to 72.2% in the second survey, and 91.5% in the third survey, after bednets were distributed. Of those that washed their bednets in the first survey, 64.9% did so because they smelt, mainly of urine from babies. 28.8% washed their nets because

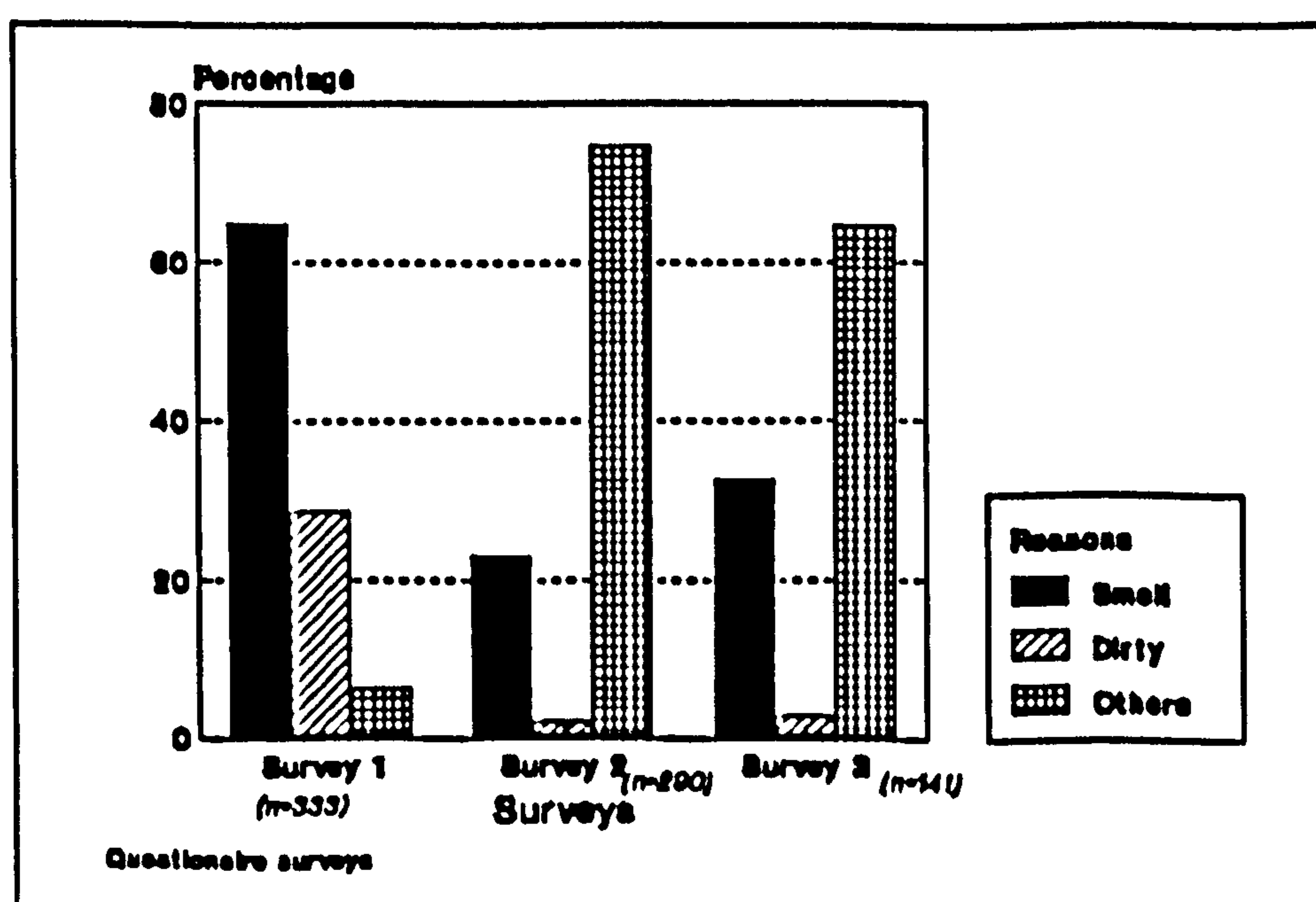


**Fig.94:**      *The frequency of washing bednets.*



they were dirty (Fig.95). Those who washed their nets because they smelt were, 23.1% in the second survey, and 32.6% in the third survey. But the majority gave 'other' reason for washing their nets. This included those who washed their nets annually, before the nets were re-impregnated, after being advised to do so. Even though some claimed to have washed their nets

**Fig.95:**      *Main reasons for washing bednets.*



earlier, because they were dirty, the majority of those who felt their nets were dirty, waited till they were advised to wash them. Again most of those who washed their nets earlier than advised did so because they smelt.

#### 4.5.4 Observation on Practice in the DDT Group.

In the DDT group, the proportion of selected households that showed evidence of recent DDT spray in most of the walls were, 89.3% in the first survey, 67.5% the second survey and 58.9% in the third survey. This reflected in practice, what was revealed in the questionnaires. The people had refused DDT spraying in increasing numbers. In this group, in the first survey, 15.2% of family members owned bednets (mostly cotton), 10.3% in the second survey, and 15.9% in the third survey (Table 44). On surprise visits to selected houses of those who owned

**Table 44:** Household members with bednets, house sprayed with DDT, and time members entered inside houses and bednets, in the DDT Group.

OBSERVATIONS IN DDT GROUP				
		Survey 1	Survey 2	Survey 3
House Sprayed (%)		89.3	67.5	58.9
Own/share bednets (%)		15.1	10.3	15.9
Under bednet by (%)	8 pm	61.5		
	9 pm	71.2		
	10 pm	92.0		
Inside house by (%)	8 pm	56.2	51.3	66.6
	9 pm	92.4	89.9	91.1
	10 pm	98.7	97.0	98.5

bednets in the first survey, 61.5% were under them by 2000 hours, 71.2% by 2100 hours, and 92.0% by 2200 hours. Those who did not get under the nets by this time were mainly mothers or



fathers who shared nets with their young children. This high compliance was because most of the bednets were for babies and young children who went to sleep earlier. The remaining member that did not get under the nets were either mother, or father if he shared the net with his wife and baby. This inspection was not repeated in the second and third surveys as households members were not keen to participate.

Similar surprise visits in selected households revealed that over 50% were inside the houses by 2000 hours in all surveys (Table 44). Similarly by 2100 hours about 90% were inside and by 2200 hours almost all household members were inside. These proportions included those already inside bednets in the first survey. It was mainly older males that remained outside their houses. There were little variations in these results between surveys. But it meant that if the shelters were sprayed with DDT, at least more than half of the household members would be protected, inside the house, by 2000 hours. This is provided the vector is killed by DDT and it bites indoors later than 2000 hours.

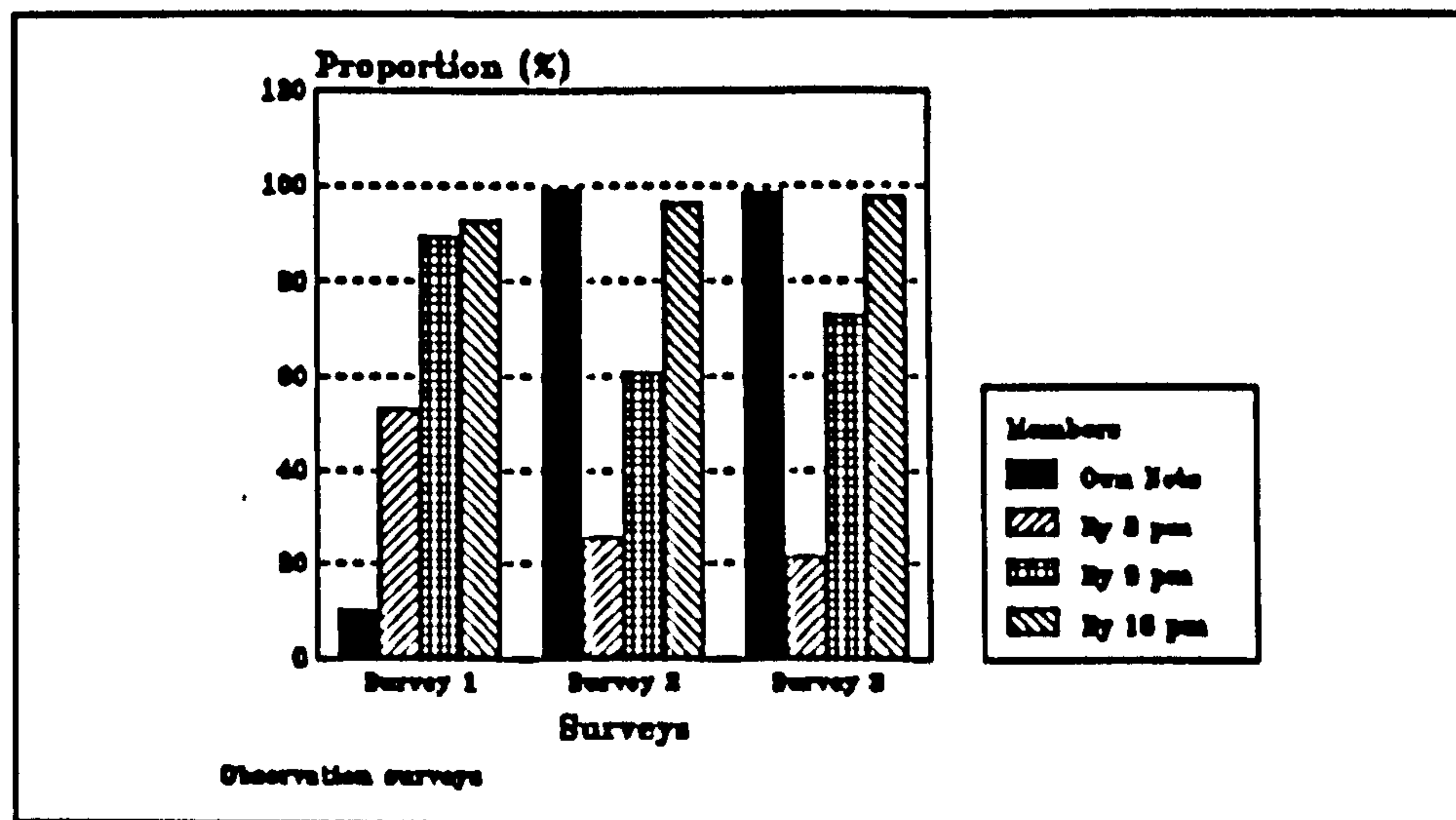
#### **4.5.5 Observation on Practice in Bednets Group**

On observation in the selected households in the bednets group, 10.1% owned a bednet, mostly cotton, in the first survey. These were mostly babies and mothers who shared the nets with them. As expected, bednets ownership increased to 98.9% in the second survey, and 98.5% in the third survey. Those who did not have any nets were missed out in the original distribution.

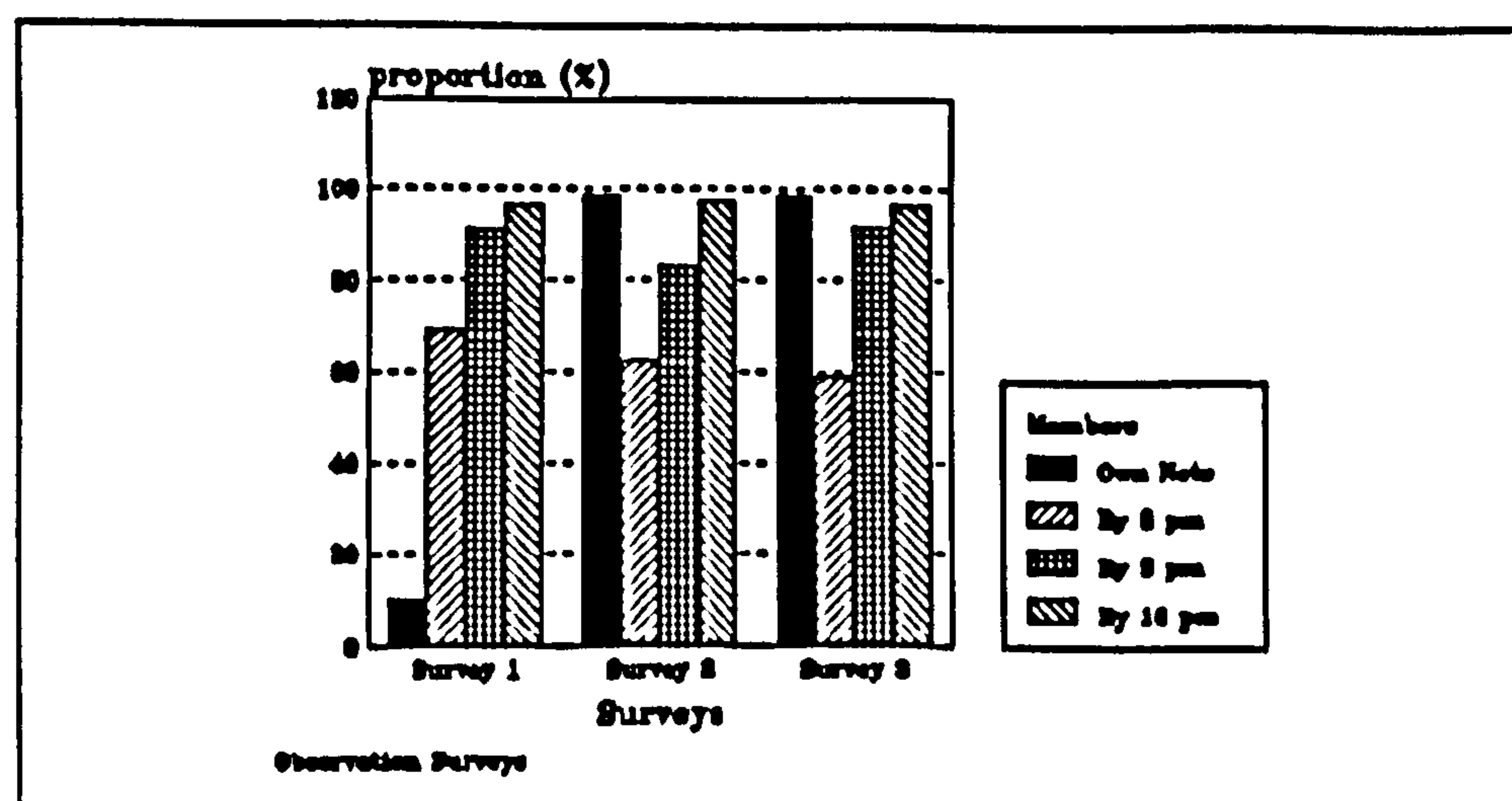
On the surprise visit to the houses in the first survey 53.2% of those who owned nets were under them by 2000 hours, by 2100 hours this had increased to 89.5%, and by 2200 hours to 92.6% (Fig.96). Those who remained outside the nets were either mothers or fathers who shared the nets with their children. In the second survey however, only 25.6% were under the nets by 2000 hours, and 21.4% in the third survey. By 2100 in the second survey 61.1% were



**Fig.96:** *Time members get under bed nets in Bed Nets group.*



**Fig.97:** *Time members get inside houses with treated bed nets.*



under the nets, and 72.8% in the third survey. But as with the first survey over 90% were under the bednet by 2200 hours. Those that did not get under the nets by this time, were mainly male adults.

At the surprise visits 69.4% were inside the house by 2000 hours in the first survey, 62.4% in the second survey and 59% in the third survey. Those that were not inside the house at this time were mainly young adolescents, or adult males. By 2100 hours 91.2% were inside their houses in the first survey, 83.2% in the second survey and 91.7% in the third survey. Over

95% of household members were inside the houses by 2200 hours in all surveys (96.9% in first survey, 97.5% in second survey and 96.8% in the third survey) (Fig.97). It was the adult males who were not indoor, and were either engaged in night fishing, or went out of their homes to meet with other villagers.

## **4.6 COMPARATIVE COST ANALYSIS.**

### **4.6.1 Comparative Costs of DDT Spraying and Permethrin Impregnated Bednets Operations**

The study took place in Florida Islands, between July 1986 and December 1989 covering a total population of 7712. The group of islands are twenty miles north of Honiara, thus in comparison to the rest of the study communities, the cost in providing support from central authority for services in the islands was somewhat moderately expensive.

During the study the following data on operation, management and materials were collected with their costs; personnel, salary and wages, transport equipment, fuel and oil, spare parts, repairs and maintenance, other facilities, equipment and materials, and the total time taken per cycle of operation. The first year DDT indoor spraying was carried out as usual and all quantifiable data needed for DDT spraying operation were obtained for two spraying cycles, July 1986 and February 1987. Then in October 1987 permethrin-impregnated mosquito nets were distributed to the same population for another separate field trial which provided an excellent opportunity for comparable cost analysis.

The data collected are shown in Table 45 for DDT spraying operations and Table 46 for permethrin-impregnated bednets. The capital items once bought with capital funds were useful for a number of years during their productive lifespan, but throughout these years their efficiency was gradually reduced by wear and tear which created costs and reduced performance value. They were annualised based on similar calculations adopted by Phillips (1987) in costing clinic



services.

The annualization process in brief consisted of determining the current value of an item (capital cost at beginning of project) and the expected years of useful life, from manufacturer of the item and the discount rate from national financial institutions. Using the latter two variables the annualization factor can be obtained from the table provided (Phillips, 1987)

**Table 45. Average Cost of DDT House Spraying per cycle in Solomon Island dollars (US\$1=SI\$3.20).**

ITEM	COST/ITEM	AMOUNT USED	TOTAL COST
<b>DDT</b>			
75% d.w.p.	\$11.50/kg	978.8 kg	\$11,256.20
20% EC	\$12.10/ltr	245.20 litres	\$ 2,966.90
<b>Transport*</b>			
<b>Equipment</b>			
OBM**	\$876.30	2 x 20 weeks	\$674.12
Canoe	\$619.22	2 x 20 weeks	\$476.32
Spray pump	\$20.34	8 x 20 weeks	\$ 63.59
<b>Operations</b>			
<b>Salary</b>			
Leader	\$601.00/month	1 x 20 weeks	\$3,005.00
Technician	\$441.40/ "	2 x 20 weeks	\$4,414.00
Spraymen	\$100.00/ "	6 x 20 weeks	\$6,000.00
Driver	\$200.00/ "	2 x 20 weeks	\$2,000.00
Fuel/oil	85c/litre	800 litres	\$680.00
Spares/ stationery/ Maintenance			\$1,328.00
<b>TOTAL</b>			<b>\$32,864.13</b>

\* Total cost annualised to cost per productive year of life.

\*\* OBM - Outboard Motor engines



which is also in *Lotus 123* computer programme package. The annualized cost, cost per year of useful life of the item, was finally determined in dividing the current value by annualization factor, which for capital items involved in this study is summarised in Table 47.

**Table 46: Average Cost of Permethrin-impregnated Bednet operations per cycle in Solomon Islands Dollars (US\$1=SI\$3.20)**

ITEM	COST/ITEM	AMOUNT USED	TOTAL COST
<b>Bednets</b>			
Single	\$10.00	4233	\$42,330.00
Double	\$20.00	1705	\$34,430.00
<b>Permethrin</b> (S - 9 mls D - 16 mls)	\$96/litre 50%EC	65,377 mls	\$6,276.20
<b>Transport*</b> <b>Equipment</b>			
OBM**	\$876.30	2 x 3 weeks	\$101.12
Canoe	\$619.22	2 x 3 weeks	\$ 71.45
<b>Operations</b>			
<b>Salary</b>			
Leader	\$601.00/month	2 x 3 weeks	\$ 901.50
Technician	\$441.40/ "	4 x 3 weeks	\$1,324.20
Driver	\$200.00/ "	2 x 3 weeks	\$ 300.00
<b>Fuel/oil</b>	85c/litre	800 litres	\$340.00
<b>Spares/ stationery/ Maintenance</b>			\$150.00
<b>Impregnation equipment</b>			\$100.00
<b>TOTAL</b>			<b>\$85,994.47</b>

\* S-9mls, 9 mls required per single net,  
and D-16mls, required per double net.

\*\* Total cost annualized to cost per productive year of life.

One cycle of DDT intradomicillary spraying operation for this population cost on average a total of \$32,864.13. But there were two cycles in a year resulting in a total annual cost of \$65,728.26 as most of the expenses would recur apart from transport equipment and spray pumps. Thus based on the 1989/90 costs the DDT spraying operation for Florida Islands were \$8.53 per person per year.

Similarly the whole operation for permethrin-impregnated bednets, with high initial capital costs for bednets, totalled a staggering \$85,994.47 in the first year, which resulted in a cost of \$11.15 per person per year. But each bednet was expected to last at least five years, which many of them actually did. When their cost was similarly annualized, the total cost of one cycle of operation was actually \$29,275.37. Recurrent operational expenses were met once a year on re-impregnation of the already distributed bednets. Therefore the average cost of using permethrin-impregnated bednets in this population was \$3.85 per capita per year.

**Table 47: Annualized Cost of main Capital items in the study in Solomon Islands Dollars.**

ITEMS	VALUE (COST)	USEFUL LIFE	DISCOUNT RATE	A.FACTOR	ANNUALIZED COST
Bednets					
Single	\$10.00	5 yrs	10	3.791	\$2.64
Double	\$20.00	5 yrs	10	3.791	\$5.28
OBM	\$2,179.50	3 yrs	10	2.487	\$876.35
Engine					
Canoe	\$2,347.00	5 yrs	10	3.791	\$619.22
Spray					
pumps	\$125.00	10 yrs	10	6.145	\$20.24



#### 4.6.2 THE COST BENEFIT OF PCD MECHANISM

##### a Mean cost of processing a blood slide.

The data collection, as explained was done in two parts. First the costs of laboratory procedures were recorded. The cost of Giemsa powder, buffer salts, alcohol and pure glycerol to make Giemsa stock, distilled water and buffer salts to mix with stock to make Giemsa stain. The cost of glass slides, microscopes, and laboratory equipments were also obtained and where necessary annualized to years of useful life. Then the salaries of the microscopists working in the area of study were obtained and mean fortnight salary of a microscopist was calculated. Since the staining was mainly done by the microscopist themselves, it is included in the fortnight salary of a microscopist. These were calculated to the mean cost per fortnight, as in Table 48.

The number of slides examined each day, randomly chosen in the selected laboratories,

**Table 48: Mean cost (SI\$) of processing and examining malaria blood slides per fortnight.**

MATERIAL	COST/UNIT	AMT. USED PER FORTNIGHT	MEAN COST PER FORTNIGHT
Giemsa stain*	\$4.24/litre	3,500mls	\$29.30
Staining equipment**	\$0.25 per fortnight	2 weeks	\$0.25
Microscope** (Binocular, Olympus)	\$823.59 per year	2 weeks	\$31.68
Glass slides	\$0.08 each	1000	\$80.00
Microscopist (+stainer)	\$249.09 per fortnight	2 weeks	\$249.09
TOTAL			\$390.32

\* included costs of Giemsa powder, methanol, pure glycerol, oil buffer salts, and distilled water.

\*\* Annualised to cost per productive year of life.

was totalled and gave the mean number of blood slides examined per day to be 98.5 blood slides



per microscopist who worked in an average of 8 hours a day and five days a week. This meant a mean number of 985 blood slides per microscopist per fortnight. When all operational costs are taken into account as in Table 47 the average direct cost of examining each blood slide is \$0.40. This cost did not include time of health worker making smear, transportation of slides to laboratory, results to health worker and capital cost of laboratories.

**b. Mean interval for blood slides to be examined.**

For the second part of the study the randomly selected, weighted average of blood slides examined from the following laboratories were obtained, Gizo and Munda serving the study areas in Western Province, and Tataba and Buala which served the study areas in Isabel Province. The average mean time taken from the date the blood smear was made, to it being examined was recorded. This was used to estimate the mean interval between taking the blood slide from the patient to the result being received by the health worker who manages that patient. The results are in Table 49.

**Table 49: Mean time (Days) taken by malaria PCD blood smears to be made and it being examined, in four malaria laboratories.**

LABORATORY	OBSERVATIONS	MEAN	STANDARD ERROR	95% C.I
GIZO	376	4.4	0.22	3.97-4.83
MUNDA	202	6.3	0.39	5.54-7.06
TATABA	131	3.1	0.31	2.49-3.71
BUALA	191	11.4	0.59	10.24-12.56
TOTAL	N=900	6.1	0.21	5.71-6.53

In weighted samples of 900 blood slides, it took a mean time of 6.1 days for blood slides to be examined. With a standard error of 0.21 the true mean lay within the 95% confidence

limits of 5.71 days to 6.53 days. However in the samples there were blood slides that were taken and read within a day (20.5%) as they were mostly taken from the same post as the laboratory. When these were excluded the mean time came to 7.7 days (standard error 0.23, and 95% C.I. 7.25 days to 8.15 days). This did not make much difference as a lot of blood smears (79.50%) came from outside the post where the laboratories were situated. The mean time however varied from laboratory to laboratory, lowest in Tatamba to highest in Buala.

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## CHAPTER FIVE

### DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

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#### 5.1 METHODOLOGY

Methodological problems in measuring the impact of disease interventions have been recognised (Blum and Feachem, 1983). There have been difficulties encountered by several studies and at times methodological flaws were alleged to have been committed, in several intervention, trial including those evaluating the impact of insecticide impregnated bednets (Bermejo and Veeken, 1992). These difficulties have all been carefully studied and looked at in this study which compares the community impact of permethrin treated bednets and DDT residual house spraying.

One main point was the unit of study which was to be randomised. Several studies randomised individual members within communities to evaluate an intervention which is aimed at an overall community impact, such as malaria intervention studies. However it is the community that should be the unit of study in such a trial and the variables measured should be compared as variation between community summary values rather than variations between individuals (Smith, 1987). Therefore the problem with some studies was that they were actually samples of one to one comparison, exposed to several problems of variations in prevalence and incidence over time, between locations and populations. This created the dilemma of how many communities can be randomised to provide an adequate sample size, but which could be operationally feasible within available resources and time. This comparative intervention study minimised these difficulties by randomising fourteen communities, into seven pairs and this gave



it a high power of study (95%) calculated on the pre-intervention survey results.

Variation of malaria prevalence and incidence within communities, between communities and even between populations within communities is another important methodological problem. These variations are due to factors of which seasons, population movements and occupations are most important. No two communities are homogeneous and without confounding variables posited by such heterogenic factors. This further makes it difficult to have an adequately ideal comparison area. The classical design to minimise confounding variables by randomised case control study is not feasible in this intervention study because communities interact and cannot be separated by real boundaries that statisticians like. However it was designed so that these problems are minimised by sampling "pairs" of communities that are as similar as possible. The communities are followed up at equal periods of time according to the intervention as the dependent variable, irrespective of seasons and other factors.

Appropriate health indicators, as the outcome to be measured for evaluation, was another difficulty. Several indicators have been adopted at length in intervention studies, with variations in definition, such as morbidity, mortality, episodes of fever, and parasite density (Snow, *et al* 1987). In this study the main measured variables are malaria prevalence and incidence, clearly defined. These variables have been those traditionally used and can be reproduced, to compare other similar intervention studies.

Intervention studies which include interviews and questionnaires often encounter the problem of accurate recall, as well as lengthy interviews which would exhaust both interviewee and interviewer. Social surveys are most important in cost-effective ways to evaluate impact of interventions and when properly designed and carried out would provide valuable rapid assessment of the impact of interventions (Manderson and Aaby, 1992). In this intervention trial, these difficulties are minimised by careful selection, structuring and proper pretesting of

questionnaires, careful structuring of variables for observation, and asking only a few basic questions that are essential and most important for the study (Fine, P. - personal communication, 1988). The questions measure the current knowledge and practice, do not require lengthy recall and would neither exhaust the interviewee nor the interviewer.

Another difficulty was the quality of tests adopted to measure the outcome, be this laboratory test, or defined yardsticks for measurements, in terms of sensitivity and specificity. This was important in a comparison trial which needs to measure the relative difference between two or more interventions (Smith and Morrow, 1991). It was necessary that the test was highly sensitive (and with high specificity). When this was so the relative rate,  $R$ , of the observed proportions in interventions, impregnated bednets ( $r_1$ ), and DDT house spraying ( $r_2$ ), would be correct representatives of the true relative rate ( $R=r_1/r_2$ ) between the interventions. The difference ( $D$ ) in their observed proportions (ie.  $D = r_1 - r_2$ ) would represent the true difference. This difference would be the proportion protected by impregnated bednets over that of DDT. But if sensitivity was low (and specificity remains high) only a proportion ( $k$ ) of true positives in both intervention areas would be detected. The relative rate between intervention ( $R = kr_1/kr_2$ ) would still be a good estimate of the true relative rate between interventions, provided that the sample size was adequate, but the observed difference ( $D=kr_1-kr_2$ ) would be much less than the true difference. This may not matter much as the relative rate would still be significant and could be further improved by increasing the sample to increase the study power. The prevented fraction by impregnated bednets would be much less than the true value in this situation.

Disastrous results would occur if the specificity of the test was low. This will lead to detection of a proportion of false positives, ( $s$ ). In this situation the observed difference between the interventions  $[(r_1 + s) - (r_2 + s) = D]$  would still be a good representation of the true difference. The relative rate observed  $[R = (r_1+s)/(r_2+s)]$  between intervention would be less than



the true relative rate and be biased towards zero. This would make the study non-significant and increasing the sample size would not improve the result. In this comparative study these difficulties were minimised by adopting the laboratory microscopic examination technique as the main test for the principal variables. The test was adequately sensitive and specific and was further improved by careful cross checking, maintenance of other quality control measures and increasing sample size from the originally calculated four pairs of communities to seven. This raised the sample size and study power.

The correct statistical technique to make inference and significance tests was important when the unit of study was a community and sample size small. When communities are randomised, sampled and compared, the sample size was often so small it could not be corrected by suitable transformation for traditional parametric statistical tests, such as *t-test*. At the same time there is obvious non-normality in such small samples of communities. Therefore the correct statistical technique in this situation and which was adopted in this study was a non-parametric statistical technique (*Wilcoxon Rank Sum Test*) which tests the significance between variation in community summary variables.

This large scale community based comparison trial between permethrin impregnated bednets and DDT spraying has been carefully designed to be as epidemiologically and scientifically a community intervention trial as possible (Kirkwood and Morrow, 1989). The heterogenic factors that could confound the outcomes were minimised. Thus the positive impact of permethrin impregnated bednets over DDT spraying was an epidemiologically sound and valid result in the prevailing situation in Solomon Islands. The conclusions that were made are therefore reliable, comparable, and reproducible by similar studies with proper careful designs which take into account the above important methodological factors.



## 5.2 THE MALARIA PROBLEM AND ITS EPIDEMIOLOGY.

The Solomon Islands had evolved through several years of antimalaria activities from eradication to control, in an effort to develop malaria control within primary health care. It was necessary to specifically and explicitly identify what would be effective, affordable and feasible within that lowest level of health care. Throughout these changes all evaluation methods were stopped except PCD mechanism, because of its epidemiological and clinical necessity. The funding of the programme became a burden with limited resources (Abel-Smith, 1988). The management of malaria was with "clinical treatment" which was basically the radical treatment of P.falciparum. The lengthy radical chemotherapy for other species, and the classical 'presumptive' treatment whilst waiting for laboratory result, ceased. The problem with increasing drug resistance with P.falciparum continued, and there was the possible emergence of P.vivax resistance which was increasingly reported in the neighbouring Papua New Guinea islands (Schuurkamp, *et al* 1992). These problems placed a tremendous pressure on costs, requiring expensive drugs, repeated clinical management of relapses with P.vivax and recrudescence of drug resistance strains. The primary health care services were faced with difficulties of effectively managing malaria with second line drugs. Such pressure would limit the scarce available resources in primary health care to benefit all those who needed them most (Forster, 1991). Therefore the urgency to identify an appropriate vector control method, even in primary health care, had been more urgent than at the present time.

Even though the endemicity of malaria in a classical sense was mesoendemic, there were enormous variation between and within islands, regions and communities and also over time. The communities were currently experiencing an epidemiological transition with malaria problems, emerging from years of low level transmission (Avery, 1974) to that of increasing malaria transmission (Kere, 1987). Even though the average parasite index (API) was about 25%, there

were variations in API in many communities. For example, in communities where DDT spraying refusals had been high, such as Malaita, Guadalcanal and Florida islands in this study the API was between 35% and 50%. In other area where DDT spraying had been effective till lately, epidemics had stabilised into high endemicity with API of 25% to 35%. Examples of such places were Kolobangara and northeast Vella La Vella. In some communities, such as Isabel island, the increase started at the time of the study in the late eighties, to an of API 20% to 30%. There were communities that did not experience high transmission till the early nineties. Such examples were, Vona Vona and northwest Vella La Vella, who experienced API between 15% and 20% with a higher API late on in the study in the DDT area.

These epidemiological transitions from low to high transmission had important consequences on the communities that were affected. There were several outbreaks in many areas affecting several people at a point in time, initially with P.vivax, as seen in Vona Vona and northwest of Vella La Vella, but these quickly progressed to higher levels of P.falciparum, as in Kolobangara and the bednet area of Isabel island. Severe malaria was affecting all ages but especially young adults and children who did not previously come into contact with the antigens. They had not a chance to develop immunity against them. The large susceptible population during the period of low endemicity was contributing to outbreaks in many communities. It was theoretically suggested (Christine Rzecypeck 1991 - personal communication, with a current collaborative research between QIMR and SIMTRI, Lines and Armstrong, 1992) that the outbreaks might have been caused by the same parasite species but of a different strain from that previously encountered.

The consequence of the transition was the increasing burden of malaria on the communities and programme that tried to control it. Even though an improved reporting of deaths was still desired, malaria was the sixth main cause of death in all ages, and might have



significantly contributed to a higher proportion of childhood mortality, since it was one of the most important public health diseases affecting the young population in Solomon Islands. This would be a subject of future research, even though childhood mortality was not as high as in the Sub-Saharan Africa. The adverse impact malaria has on the population was tremendous. Whilst available funds were decreasing (MOF, 1991, Abel-Smith, 1986), the cost of traditional malaria operations were increasing (Mills, 1991, Phillips and Mills, 1991) with resulting adverse impact on the life, social welfare and income generating ability of the family (Mills, 1989). The epidemiological situation in which malaria was experienced, with increasing drug resistance (including new ones), the high cost of drugs (Bjorkman, 1991), including delivery (Forster, 1991) the difficulties with effective chemoprophylaxis programme (Allen, *et al* 1990) and the unlikelihood that an effective vaccine would soon be available (Mendis, 1991, Chefas, 1990) were important facts. If and when an effective vaccine is available it would still have to find its way to those most in need of it (Targett, 1991, Hall, *et al* 1990, Blood, 1989). There was no best alternative available but to continue pursuing appropriate vector control intervention. This is the main option available to address malaria as the public health problems of communities. The results on this permethrin impregnated bednets and DDT residual spraying comparative trial were encouraging particularly on the various different epidemiological variations in endemicity that were identified.

### **5.3 THE COMPARATIVE IMPACT OF THE INTERVENTIONS.**

#### **5.3.1 On the Prevalence of Malaria Infection**

The results and interpretation of the study that permethrin impregnated bednets were more effective than DDT in reducing and controlling the prevalence of malaria in the study communities. In general bednets prevented an estimated 41.08% of malaria relative to DDT



residual spraying. The reduction was particularly dramatic in young children, especially infants, where complete protection was possible. The reduction in P.falciparum, which was very encouraging, was the species that was responsible for mortality due to malaria (Greenwood, *et al* 1987) and had been revealing its adaptability and plasticity against antimalarials.

The relative protection by permethrin impregnated bednets was also much higher in the under 10 year olds, with at least 50% of the expected prevalence that would have occurred with DDT (Table 24). Infants have the highest protection rate, with older children (10-14 yrs) the lowest. The prevented fractions in all ages were significant and in all cases markedly so with P.falciparum. The species that predominated was P.vivax especially, in the older age groups of 10 to 14 year olds. Even though the reduction was still significant in adults the ratio between P.falciparum and P.vivax did not differ greatly.

The successful reduction of P.falciparum with impregnated bednets confirmed that many vectors were killed before the sporogonic cycle of this parasite species was completed. This was also supported by the decline in parous rate with impregnated bednet (Table 41). The precise impact of impregnated bednets on the survival rate of the vector has yet to be measured by careful examination of ovarian dilatations of an adequate sample, but recent studies (Magesa, *et al* 1991) seemed to indicate that the survival rate was probably reduced with bednets as it was with A.gambiae. There were mosquitoes which may have missed impregnated bednets and continued to remain outside transmitting malaria even at a low level, especially on some older people who wandered outdoors (as was observed in villages surveyed).

These results also showed that DDT spraying failed to decrease malaria prevalence and was unable to prevent it increasing. In the epidemiological situations in the study communities, with the evolving resistance of both vectors and communities (both behavioural), DDT spraying was no longer effective against the prevalence of malaria infection. The finding that permethrin

impregnated bednets decreased malaria prevalence better than DDT, regardless of variations in prevalence existing in the study area, was important. However the prevalence would not decrease further than, about 10%, to make it a potential eradication method. Thus in the current approach, permethrin impregnated bednets could not reduce the prevalence much lower, where transmission of a steady low number of P.vivax and P.falciparum continued, especially in the older age group. At this stage permethrin impregnated bednets would need an appropriate complementary measure, which might depend on a careful strategic approach to different epidemiological situations.

Permethrin impregnated bednets as a malaria control intervention were quite successful in reducing prevalence and efficiently maintaining it at a low level with little variations according to seasons, population mobility and occupation that would affect transmission. The standard deviations of the means with impregnated bednets were significantly smaller than those of DDT at all ages. Prevalence varied much in the DDT group, with larger standard deviations. This suggested that DDT was unable to contain malaria transmission when affected by factors such as rainfall, population movements and occupation.

This important degree on the impact of permethrin impregnated bednets on malaria prevalence was not experienced in many studies elsewhere, all of which nevertheless concluded that permethrin impregnated bednets (Rozendaal and Curtis, 1989) would be effective in situations where malaria endemicity was low. This would be quite an important factor in the study communities. Differences would also be related to how the impacts were measured and difference in the definition on which the measurement were being made. Permethrin impregnated bednets did not have any significant impact on malaria prevalence in the Gambia (Snow, *et al* 1987), and differences in malaria prevalence between bednets and DDT house spraying were less convincing in Tanzania (Lymio, *et al* 1991). In Papua New Guinea (Graves, *et al* 1988) where



malaria was transmitted by the same vector as the Solomon Islands, the only significant impact on prevalence was seen in the under fives against *P.falciparum*. A similar conclusion was also made on children in Malaysia (Hii, *et al* 1987).

Why was it that permethrin impregnated bednets were significantly effective against malaria prevalence in this study? There are several possible reasons which may have importance if and when bednets are being operationalised in a control programme. One important aspect was how studies were designed, implemented and the outcomes adopted for measurement (as discussed in Section 5.1). In this study permethrin impregnated bednets were applied in seven community-wide operations aiming to minimise the risk of being infected by the vector population over a longer period of time. They aimed at reducing the infective potential of the vector population while in several other studies were targeted on villages, or families within villages, and were in effect short-term trials (Hii, *et al* 1987, Graves, *et al* 1987, Msuya and Curtis, 1991). Therefore the gametocyte pool in the whole community would still be maintained by families or villages not protected, to infect the vectors. Subsequently, even those who used impregnated nets, faced the same risk of inoculation by the same vector population, as those without nets, when they were outside their nets.

Another reason was that people found positive with malaria parasites during the prevalence surveys were treated fully every time. Therefore the prevalence rates determined, were a direct consequence of the prevailing infectivity of vector populations in response to the intervention. Since the interventions were targeted at the community, the positive cases were representatives of new infections with a direct relationship to the existing transmission level by the vector population. As such permethrin was conclusively much more effective than DDT. The different designs and application in Tanzania (Lyimo, *et al* 1991) did not find similar significant difference between the same interventions as this study. It is important however, that as with all



studies, any strategy that was adopted and found effective, would be of no use unless it can be operationalised (Mills and Bradley, 1987). This approach was adopted with the extensive PHC network that had been established, and was in place. This network would allow the early treatment of people suspected with malaria. If the infectivity of the vectors could be reasonably minimized, a suitable combined strategy was determined to deal with malaria in primary health care. The finding, therefore, in addition to explaining the significant difference with prevalence rates between interventions, was very important in that permethrin impregnated bednets would technically be an appropriate component of malaria control strategy in primary health care where effective early treatment could also be assured.

The efficiency of the infectivity of the vector population also would explain the differences between studies. A highly efficient vector would continue transmitting malaria even at very low density. The sporozoite rates of A.gambiae were reported continuously to be between 4% to 7% (Lines, *et al* 1987, Magesa, *et al* 1991) making it the most highly efficient vector to transmit malaria in man, compared with the average sporozoite rate of A.farauti of 0.64% as found in this study. A much more effective intervention that would have a bigger reduction on density, or much more important, shortening of longevity, by affecting survival rate, would be needed against A.gambiae than would be for A.farauti. A recent study in Kenya (Sexton, *et al* 1990) found a significant decrease in the density of A.gambiae and A.funestus, but those that survived were still biting and had a sporozoite rate of 6.4%. In areas of holoendemic malaria, it would not be that simple to control malaria as was seen in Tanzania (Magesa, *et al* 1991), where the survival rate of the vectors were significantly reduced, but the impact on parasitaemia were only found significant in some communities with well constructed dwellings, as in plantations. The effective impact in an area where the local vector population was not so efficient was supported by bednets impregnated with deltamethrin in China. It reduced malaria

prevalence transmitted by, A.anthropophagus (Xu Bozhao, 1992- personal communication, Li-Zu, *et al* 1989), A.dirus and A.minimus (Li-Zu, *et al* 1987, Curtis, 1992). Malaria in these areas was hypoendemic with less efficient vectors than A.gambiae.

The difference in malaria endemicity between areas where studies have been done, contributed to the observed differences. Apart from the important part played by the infectivity of the vectors, there were different transmission patterns as caused by seasonal variations, population movements, immunity levels and occupational differences. In many parts of Africa malaria was holoendemic, hyperendemic and seasonal, and this affected interventions and how and when they were evaluated. Finally these differences could also be explained by the level of compliance in the use of bednets. Even though there was extended protection, an individual can only completely benefit from it by using it properly and not washing it prior to re-impregnation. This was shown in the Gambia (MacCormack and Snow, 1986) Tanzania (Njunwa, *et al* 1991), and Malaysia (Leake, *et al* 1989) where no convincing impact was seen in those who did not use bednets well. In this trial, the community compliance rate to permethrin impregnated bednets were maintained at high level, above 80%, contributing to the effective impact on malaria prevalence and there was no significant problem with washing.

### 5.3.2 On Incidence of Malaria Infection.

Any disease intervention under trial would be of no benefit and or a bad investment, unless it has the potential to reduce that disease (Rosenfield, *et al* 1984). In this trial that disease was malaria which was causing unsurmountable burden to the tropical world, especially the poorest and underserved (Remme, 1992). But as has already been discussed, appropriate and correct outcomes had been adopted in evaluating the changes of malaria disease as a consequence of the intervention. Several definitions of malaria illness have been adopted, malaria



illness, either fever episodes, parasite density or both with other concomitant clinical signs and symptoms (Bermejo and Veeken, 1992). These variations made direct comparison difficult and in some cases they were not appropriately used. But some outcome which, as accurately as possible, provides a reliable measure of the target disease, is necessary.

The incidence of malaria infection as defined (Section 2.5.3) had an important direct correlation with clinical episodes of malaria illness. The measurement was based on blood slides taken from ill people who were suspected of malaria (PCD) and were found with the parasite. The use of this variable assumed that two important requirements had been fulfilled and there was sufficient evidence that they were (Briese, *et al* 1989, Fardy, *et al* 1990, Kere and Quan, 1992). The first was that ill people who were suspected with malaria and had their blood smear taken, received full treatment successfully. Second was the extensive primary health care network in place with several PHC workers in the field, as discussed for each study area, was adequately equipped with resources to take slides and administer early treatment. In this way a new slide positive case would represent a malaria infection contracted through the vector population against which the intervention was being applied. When this was the case, the results on incidence should, at least, show the same conclusion as on the prevalence of infection, as discussed earlier. This was also important in clearly examining how appropriate the PCD mechanism was in evaluating the malaria control intervention in primary health care.

The changes on incidence of infection with permethrin treated bednets and DDT residual spraying followed closely that of changes in the prevalence of malaria infection. Two comparisons could be made with the incidence of infection, as collection of data began in all study areas 10 to 12 months prior to bednets, and continued for another 20 or so months.

When the incidence of infection prior to bednets and after bednets were compared there were marked reductions with permethrin impregnated bednets, from an average of 66.2 cases per



1000 population to 31.39 per 1000 population in all ages. The reductions in mean incidence with bednets were highly significant, especially in the under fives ( $p < 0.0001$ ). It was also significant in other age groups at least at 5% level. The variations of the mean incidence as discussed, in Tables 27 to 37, decreased significantly after impregnated bednets were introduced. This showed that permethrin impregnated bednets decreased the incidence of malaria, and maintained it at a low level with less variation than would have been otherwise. This was especially convincingly in infants and especially with P.falciparum. It was mainly the older age groups and adults that contributed to incidence. This was a similar finding to that in Papua New Guinea (Graves, *et al* 1987) where permethrin impregnated bednets were used against the same vector of malaria.

When considering the fraction of mean incidence prevented by impregnated bednets according to specific age group as summarised in Table 50 the conclusion could be made that permethrin impregnated bednets prevented at least a half of the malaria incidence in the same

**Table 50. Prevented fraction of mean incidence of malaria with introduction of impregnated bednet in the bednet area**

AGE GROUP	Prevented Fraction in malaria incidence (%)	
	All species	<u>P.falciparum</u>
Infant	74.7	82.5
1-4 yr	55.6	66.1
5-9 yr	57.5	58.1
10-14 yr	56.5	66.8
15 yr +	45.9	55.4
All ages	52.6	63.3

communities. In infants the prevented fraction increased to three quarter of all species and over eighty percent of P.falciparum.

When the incidence in the DDT and bednet groups were compared the reductions were

also quite significant. Even though there were variations in each individual areas before introduction of bednets the incidence decreased dramatically and the low level was maintained in the impregnated bednets group. Even though the impact was significant in all age groups, it was especially marked in infants where the incidence was maintained below 5 cases per 1000 infants a month. In the DDT group even though the average incidence was lower at the start, it increased to the highest peaks at the end of the study. There were greater variations during the transmission pattern than in the bednets area.

When comparing the fraction of mean incidence prevented by permethrin impregnated bednets over DDT, it was again estimated that more than half of the incidence was prevented with impregnated bednets in all ages (Table 51). The highest was in the 1-4 year old group. The lower fraction prevented was less than expected in infants by bednets, over DDT. This was more likely to be an added effect of bednets, even untreated, that were observed during the social surveys.

**Table 51.** The mean monthly incidence (per 1000 population) in the DDT and bednet groups.

AGE GROUPS	MEAN MONTHLY INCIDENCE		PREVENTED FRACTION (%)
	DDT GROUP	BEDNET GROUP	
Infants	13.0	5.0	61.5
1-4 yr	42.5	10.1	76.3
5-9 yr	46.9	15.4	67.57
10-14 yr	49.0	20.8	57.57
15 yr +	40.32	16.21	59.80
All ages	42.50	15.1	64.47

The resurgence of cases near the end of the study in the DDT group could be due mainly to two factors. One was that when the communities in the DDT group realised that permethrin



impregnated bednets were much more effective than the DDT spraying, which had been going on for over 30 years, their refusals became increasingly high. Only half of the respondents surveyed allowed their houses to be sprayed which corresponded very well with the observation that only 58.8% of houses inspected had some evidence of recent DDT spraying. Whatever technical protection DDT might have had on those communities was lost with increasing refusals. In order that DDT offered any protection the coverage should be at least over 80% (Pampana, 1969). Secondly, there was the increasing behavioral resistance of the malaria vector as discussed. After over 30 years of being subjected to DDT A. farauti had clearly avoided DDT spraying (Table 39) and subsequently continued transmissions despite DDT. There were two possible mechanisms in this regard as the intensive study on the vector were not carried out in all communities. One was that the vector population in many communities eventually became resistant, some later than others, as was probable in the study communities. The other was that the same strain observed to have developed resistance in some communities gradually invaded other areas and replaced the local vector population which were still sensitive to DDT. When this resistant strain population was established in the new environment, outbreaks and upsurges of malaria occurred. In this study, exactly what entomological mechanism was responsible was uncertain. Both human and vector resistance as discussed were likely to be involved and further entomological study was needed to clearly determine what part each was contributing to such late increases.

These results showed that permethrin impregnated bednets were more effective than DDT residual spraying to reduce and control malaria incidence in the study communities. This was much more marked in young children especially infants, and in particular with P. falciparum. DDT residual spraying was unable to effectively control malaria as bednets.

Bednet studies have shown varying results on malaria with several having an



unconvincing effect in reducing incidence. Even though there were some differences in definitions of incidence, almost all of these studies from Sub-Saharan Africa where malaria is holoendemic or hyperendemic, impregnated bednets did not show such significant impact on malaria incidence. One study did however have a marked effect on childhood mortality (Alonso, *et al* 1991). In Kenya, in an area of holoendemic malaria, it was recently found (Sexton, *et al* 1990) that permethrin impregnated bednets only significantly reduced the incidence of P.falciparum. This was also the case with impregnated curtains. There was no appreciable changes in incidence in Tanzania (Msuya and Curtis, 1991) where malaria was holoendemic. In Burkina Faso (Pietra, *et al* 1991) permethrin impregnated bednets were only effective during the dry season, when transmission was low.

Again studies in China (Lin-Bao, 1991, Curtis, 1992, Li-Zu, *et al* 1989) found significant reductions in malaria incidence with deltamethrin impregnated bednet, and Malaysia (Hii, *et al* 1987) and Papua New Guinea (Graves, *et al* 1987) only on children against P.falciparum. The results in those studies showed a more convincing decrease than the African studies for reasons that were discussed earlier.

Few comparative studies have been done on the impact of permethrin impregnated bednets and DDT spraying on malaria incidence and prevalence. One study in Tanzania (Lyimo, *et al* 1991, Msuya and Curtis, 1991) where DDT residual spraying was compared with permethrin impregnated bednets found that both interventions significantly reduce incidence when compared to a comparison area. There was no significant differences between the interventions even though the effect of impregnated bednets was longer lasting than that of DDT in preventing malaria incidence. The vectors of the study communities, A.gambiae and A.funestus, had not been subjected to over 30 years of DDT spraying as A.farnuti in this trial. In Malaysia (Loong, *et al* 1986) only compared bednets impregnated with both insecticides and

did find some difference in favour of permethrin impregnation. The most successful results were from China (Curtis, 1992, Lin-bao, 1991) where deltamethrin impregnated bednets were significantly more successful in reducing the incidence than DDT spraying in communities. In Hubei, China, (Xu, 1992 - personal communication) where hypoendemic malaria was transmitted by A.anthropophagus there was no difference between the DDT and impregnated bednets, but since impregnated bednets were more effective against the vector and cheaper, it was adopted as the main intervention. It was important to note that in China with an extensively well established PHC system, early treatment of clinical malaria are easily available.

The endemicity of malaria in Solomon Islands is mesoendemic and, as seen in the study, permethrin impregnated bednets successfully reduced malaria incidence and prevalence. DDT residual spraying has no significant impact on these variables. The presence of a well developed primary health care service is argued to be an important factor contributing to these favourable results.

#### 5.4 IMPACT ON THE VECTORS.

In this study permethrin impregnated bednets had a more important impact on the vectors than DDT spraying. Both interventions, especially permethrin impregnated bednets, reduced A.punctulatus to such a low level that it was not possible to continue monitoring anymore. Even so the impact on the main vector, A.farauti was very impressive, which entomologically explained the effect the interventions had on malaria.

Permethrin impregnated bednets reduced the biting density of A.farauti by 60% outdoors and 55.6% indoors, when compared with the biting density in the DDT area. The density in the DDT area, both outdoor and indoor, did not differ from that in the comparison area where no interventions were applied. Therefore permethrin impregnated bednets killed the vector and



reduced the overall density. However since the ration of indoor and outdoor biting remained similar, as with that of the DDT area, the dosage of permethrin used in this study did not create more deterreny than DDT. Recently it was observed that with a higher the dosage of permethrin (Carnevale, *et al* 1992) the repellent effect overcame the killing effect, and vice versa with lower dosage. It was therefore likely that at the dosage adopted, the decreased density of A.farauti was predominantly due to its killing effect. Such impact of permethrin impregnated bednets would be beneficial both to users and non-users in the same location. A similar reduction was also found with A.gambiae and A.funestus (Sexton, *et al* 1990) in Kenya with the same dose of permethrin, but those vectors that were still biting had high sporozoite rates.

The most striking feature with A.farauti was that it completely avoided resting on indoor surfaces. It became completely ~~e~~<sup>ex</sup>ophilic, even though still endophagic. Permethrin inhibited feeding by 47.5% which was much lower than expected, when compared to other studies that reached higher levels, as high as a 91% inhibition with A.gambiae (Lindsay, *et al*, 1991). It was therefore likely that people might have been bitten as they went out during the night. However, since these studies were made on experimental exit trap huts, mosquitoes that fed elsewhere might have entered the hut and were caught in the traps. Even though it would be possible to distinguish human blood from animal blood by established immunological techniques, to distinguish blood between humans requires a more complex method such as the PCR technique (Gokool, *et al* 1992). The killing effect was confirmed with at least a 97% mortality. There was no difference with these variables which were low both in the DDT and comparison areas. The results on bioassay however showed that both insecticides were still lethal in their applied dosage, even though there was a higher mortality with permethrin. The vector survived DDT as it did not come into contact.

These findings explained how permethrin impregnated bednets were effective against



A.farauti with such a behaviour as was described elsewhere (Curtis, 1990). The vector might miss the walls after feeding, but cannot avoid touching the impregnated nets if it had to feed. At the same time the odour produced by humans under the nets would be an attractant for the vectors to come into contact with treated materials.

The mean parity of A.farauti was decreased by 10% with permethrin impregnated bednets, a slightly larger reduction indoor than outdoor. Parity in the DDT area, which was above 53%, was not much different from the comparison area. There was a tendency for the mean parity to be higher indoors, implying that a higher proportion of transmissions were taking place indoors. Permethrin impregnated bednets were therefore suitable in this situation, even though there were still many parous mosquitoes biting outside to infect anyone who wandered outdoors. The reduction with parity was the same both indoor and outdoor indicating that impregnated bednets killed all who come into contact and both parous and nulliparous mosquitoes entered to bite. Thus entomologically, permethrin impregnated bednets were effective and would bring down malaria, but only if people use them widely and the vectors came into contact with them.

Unfortunately, with a small sample of vectors from the bednet area, the absence of sporozoite in caught mosquitoes might still be not adequate upon which to draw conclusions, but the average sporozoite rates in the DDT area (0.60%) and comparison area (0.69%) did not differ much. However in the DDT area, the indoor sporozoite rate (1.41%) was much higher than the indoor rate in the comparison area (0.65%), with a tendency to be P.falciparum. This would indicate that with DDT spraying a higher proportion of mosquitoes that entered to bite were parous mosquitoes, therefore were more mature and more likely to have mature sporozoites, including P.falciparum which have a longer sporogonic cycle. A vector population with such a behaviour towards an insecticide would give rise to more cases of P.falciparum. This was seen

in the DDT areas. The killing effect with permethrin impregnated bednets immediately had an impact on P.falciparum, whose sporogonic development with the A.farauti mosquito (Burkot, *et al* 1990) was much more efficient. Thus it reduced the potential for A.farauti to transmit malaria by its killing effect. The effect of permethrin impregnated bednets on the longevity of the vector population has yet to be determined as was done with A.gambiae (Magesa, *et al* 1991). But the information would be necessary as to whether this intervention were a potential eradication tool against A.farauti in the conditions prevailing in the communities. At least the current findings explained entomologically how permethrin impregnated bednets prevented malaria, especially P.falciparum.

## 5.5 KNOWLEDGE, BELIEFS AND PRACTICES.

Essentially any disease intervention needs the active participation of the communities who are supposed to benefit by it. Several mistakes were encountered in the past, in particular during the eradication times (Bradley, 1991) when communities were told to participate because what was being carried out was for their own good. This was inappropriate when adopting such interventions as bednets where human behaviour in the use of bednets was crucial to it being effective. As a malaria control intervention, it was necessary that the users got under the nets early enough and were protected before the vectors began to bite. This was easier said than done as behaviour of vectors did not necessarily comply with human behaviour (Leake and Hii, 1989), as one would have liked to the extent necessary for the effective use of bednets. However, a high level of compliance in the use of bednets was essential.

Before an intervention is accepted and used, it must be understood and the person convinced that it will provide the protection against malaria as is supposed. In the questionnaire survey, the level of knowledge of mosquitoes spreading malaria was high, over 65%, both in the



DDT and bednet groups. This was expected after 30 years of intensive antimalaria activities. There were no differences between education, sex and age in the responses, but what was important was those who believed the interventions killed the mosquitoes and prevented malaria. In the DDT group, those who continued allowing their houses to be sprayed in the initial interview, but this declined to 53.6% in the third survey. The main reason given by those who refused spraying was that it did not prevent malaria anymore. As expected only 60% of houses inspected had any evidence of recent DDT spray in the last survey.

In the bednet group, those who believed that bednets prevented malaria increased from 40% in the first survey to 63% in the last survey. Interestingly, those who used bednets because it prevented insects declined, from 50% in the first survey to 20% in the last survey. The communities eventually experienced the reduction in the incidence of malaria as actual experience was a more powerful tool in health education. There was no problem with washing of nets.

The time people got inside the houses in both the bednet and DDT groups did not differ. By 8 pm between 50% and 60% were inside the houses and by 10 pm almost everyone was inside the houses. Those who were outdoor were mainly older male children and adults who were playing in the village, visiting friends or went night fishing. The slight higher number in the bednet group that were inside the house by 8 pm in the later surveys might indicate an issue that would need to be closely watched. With bednets and the subsequent decline in biting nuisance, people especially the older children, might stay outdoor more and maintain outdoor transmission. This could particularly be in the A.punctulatus area where the density was very low. The users, with a high level of knowledge on the relationship between mosquitoes, malaria and bednets, might see no need to continue using bednets. The biting nuisance was no longer there and malarial fevers became rare, so they would stop using the nets. This would enable the



vector population to increase. In this regard continuing education would be essential.

The time users of bednets got under the nets were also observed by surprise visits and in the initial survey the rate by 8 pm was as high as those owning nets (mostly babies). The early evening rate dropped as the denominator increased, with more people owning nets in the later surveys. The rate at 9 pm, initially high at 85% dropped in the later surveys for much the same reason. But by 10 pm over 90% of the people were under the nets. Those who did not get under the nets by the respective times were mainly older children, mostly males, who were still playing around, and other adults out visiting friends, or went night fishing. The older age groups were the ones that were infected outside bednets, or outdoors.

An important observation in the DDT group was, what took them so long to realise that DDT was no longer effective, after more than 30 years of use? The compliance of these communities to DDT spraying had continuously been high. The other main factor in addition to what was described was that, with impregnated bednets, they were aware of a better and less hassling measure than DDT. The reputation of permethrin impregnated bednets was quickly disseminated, particularly its impact on nuisance bedbugs, head lice and other insects, and later malaria. The communities in the DDT group realised there was a better alternative and increasingly refused DDT spraying. This would favour a feasible extension in the use of impregnated bednets to further its benefit, particularly when people were willing to contribute to buying them. But caution should be taken for DDT too had similar dramatic effect during its early days which should continue to be borne in mind.

In conclusion, the findings in the social questionnaire/observation surveys clearly favoured the positive impact of impregnated bednets and not DDT spraying. Therefore the current knowledge, beliefs and practice of the communities enabled the effective use of permethrin impregnated bednets, as found with the parasitological results.

## 5.6 THE COST OF OPERATIONS

The proper classical cost effective, cost efficient and cost benefit analyses on health interventions were not impossible, but required complex sets of data and statistical and econometric techniques. If they were improperly done, the results could lead to serious errors on decisions that might be most important (Carrin, 1984). In the real sense in health care activities, interventions were often interactive and complementary to and with each other, rather than clear alternatives (Kaewsonthi and Harding, 1984). Thus to single out the cost of one alternative in the fabric of health care activities was not easy. But with shrinking health budgets, (Abel-Smith, 1988), increasing economic problems, impediments of development (Lennox, 1991) and money has obviously spoken louder than words (Phillips, 1987) in the decision making process, it was necessary that some proper cost analysis of health intervention were made (Kere and Kere, 1992). This was most important when a choice had to be made of at least two alternatives, as in this study.

This study showed that the cost of permethrin impregnated bednet operation was \$3.85 per capita per year and DDT operation at \$8.53 per capita per year would provide health decision makers and financiers an easy alternative. But cost analysis alone at times would not necessarily end with a decision. Affordable cost would not always equate with effectiveness, even though the ambition to serve more people with a given amount of resources. A good decision was sometimes difficult as several factors had to be taken into account (Phillips and Mills, 1991). Effectiveness against the disease, acceptability by people and even the cheapest intervention might not be afforded by the community or programmes serving them. The attitude of potential donors who also ask the cost of alternatives would also need to be considered.

The study, therefore, showed that permethrin impregnated bednets was more effective than DDT spraying, well accepted by the people and even at a cost that communities might be



able to afford. The initial purchasing of bednets was expensive, but as bednets were expected to last five years, the annualized cost for each bednet would be between \$2.64 and \$5.28. This could be afforded by almost every family, or even by the malaria control programme with the current budget of \$5.40 per capita per year. Therefore permethrin impregnated bednets was not only effective and well accepted, but an affordable intervention to be adopted in malaria control in Solomon Islands.

The intervention after being found effective, affordable and applied would need to be monitored and evaluated. The methods with which evaluation was being done would cost money and would consume resources. Ideally a method of evaluation that was cheap, robust and sensitive would be necessary for an intervention that was being applied in primary health care. In this study the cost and value of blood slides taken through the established PCD mechanism was being analyzed. The results showed that for every blood slide taken, processed and examined, it cost on average \$0.40, or \$40 for every 100 slides. This was a minimum, as cost of transportation to the laboratory and laboratory spaces were not included. Even at this cost, as a component of an evaluation system, it was still expensive. The costing that was done were mainly direct and institutional costs, and not costs peripheral to the laboratories and other indirect costs. A recent analysis found that in Thailand (Etting, *et al*, 1991) institutional, urban and peripheral, cost per smear per positive case was US\$0.82, and US\$1.58 respectively. The main cost was salaries and wages in both cases as in this study. The finding of this study was smaller because other indirect costs were not included and also in particular the rate for all slides and not only for positive slides. The total cost per blood smear would be much higher if the two latter measurements were included.

One of the main purposes of a blood slide was to support the health worker to treat patient effectively with laboratory results. The study found that a blood slide took a mean of 6.1



days to be examined and double that for the results to get to the health worker. Indeed, this investment was not benefiting the patient who was managed clinically. It was also found that only 20% of blood slides taken has any chance of contributing to the treatment of patients. This also meant that 80% of the investment would not directly benefit patients. This fact should be carefully considered in using this tool to evaluate an intervention in malaria control in primary health care. However, decision could not be made on these two results alone to continue with the PCD mechanism to monitor interventions. How decisions could be made so that the tool adopted provided the adequate epidemiological information required, at an appropriate cost invested and that would not disrupt the malaria control activities, would need to be carefully examined before a decision was made. This is discussed in the next section.

## **5.7 THE COST EFFICIENT USE OF PCD SLIDES AS THE APPROPRIATE MALARIA EVALUATION TOOL**

### **5.7.1 Traditional Use.**

The use of PCD blood slides was well established in the study area as well as the rest of Solomon Islands. It was set up to serve both clinical and public health purposes. Clinical, it supported patient management, and public health, to be used to monitor and evaluate the national malaria control programme. The established practice was that everyone with fever or ill health, suspected of malaria, had their blood slides taken for microscopy. Therefore what is the point of changing what is so well established?

### **5.7.2 Study Findings**

The study, as discussed above, revealed that it cost minimum of \$0.40 to process and examine each blood slide. When this was considered with the total 352,000 blood slides examined in the Solomon Islands in 1990 (Fig.13), it came to a minimum of \$140,800.00 in total

for examining blood slides, which was a lot of resources. With the average API of 25%, the cost per positive blood slide would be much higher with the total investment. However the next concern, regardless of whether the blood slides were positive or not, was that only 20% would have any chance of benefitting patient management. This would mean that 80% of the cost (\$112,640.00 in 1990) was invested for epidemiological reasons, which at primary health care level would be an extravagant exercise that nobody in poor malarious communities could afford.

The next important finding was that each microscopist examined a mean daily number of 98.5 blood slides. This was high and risked increasing inaccurate results by an overworked microscopist. The consequence of this would further decrease the benefit of blood slides. Therefore based on these findings the value of PCD blood slides needed to be re-examined, despite it being one of the most important traditional evaluation tools in malaria control programmes. This was also further justified when efforts were being made to control malaria in primary health care. The scarce resources available in primary health care should be put to as good a use as possible, for example buying essential drugs instead of investing in some activities of less benefit.

### 5.7.3 The Options

The most radical cation would be to simply cease the PCD mechanism and save resources to be used for other more needed activities, but this would create some serious problems. First would be the absence of a most reliable measure to evaluate malaria programmes. The argument that this could be done by monitoring clinical cases of malaria, after years of attention (Basset, *et al* 1991) was still an unreliable measure. The importance of malaria, especially in children were well studied (Gilles, 1966) and so were the non-sensitivity of clinical signs and symptoms to parasitaemia (Henrickse, *et al* 1971). Several studies failed to demonstrate the exact



correlation between clinical and parasitaemic malaria (O'holohan, 1976, Stein and Gelfand, 1985). Subsequently, precise definitions of what is a clinical case, to enable standardisation of data collection, has not been well established (Mkawagile and Kihama, 1986) well enough, to provide a sensitive and reliable measure.

The next question would be what would happen to the staff, equipments and other resources which had been in place. These could be used for other health purposes, but re-orientation, re-training and renovation would incur costs. The developed infrastructure were valuable resources which should be put to better use. Finally, the programme would face serious public outcry for not serving them. It appeared that patients perceived that making blood smears provided some benefit, and ceasing it might discourage them seeking health care.

The next option would be to continue, but not in the manner that it had been deployed. The current approach was suitable for an eradication/control programme, where large amount of resources were available. This would not be the same in a malaria control programme in primary health care. Therefore the modification of the mechanism for adoption into a primary health care approach would need to satisfy the following criteria.

- i. To provide an adequate sample and to be collected in a the manner that would provide the epidemiological information required to monitor and evaluate the intervention,
- ii. to save as much resources as possible for other urgent needs, when satisfying the above,
- iii. not to discourage patients away from seeking care when they are clinically ill, and,
- iv. occupy the microscopist but allow them adequate time to spend on priority blood slides, especially those whose results would benefit patient management.



#### 5.7.4 The Modified Tool.

The suggestion which is being made is argued to fulfil the above criteria. There is no doubt that PCD blood slides will still continue to provide useful information to evaluate malaria control programmes. In this multicentre comparative trial, the age group that was highly susceptible to changes with malaria between the interventions were children. This was especially infants even though the relative impact was significant in older children. The difference was particularly significant with P.falciparum. These findings were obtained on a closely implemented and monitored prevalence surveys during the study.

The finding on changes in incidence between the interventions corresponded very well with those of prevalence. This would imply that taking blood slides of all people with fever would not provide any better epidemiological information than the most sensitive group, the children, especially infants. The value of PCD results in infants has long been recognised (Viswanathan, 1941, Macdonald, 1950, Foll, 1968, Pull and Grab, 1974) and its importance was recently reviewed (Brabin, 1991) and suggested as a simplified method (Webber, 1992) for measuring the effectiveness of malaria control intervention in primary health care. Therefore epidemiologically, the most sensible decision would be to only monitor the PCD slides of infants and leave out anyone older. Whilst this would provide a most sensitive group and save considerable resources, it would leave one with quite a small sample which might not be easy to have access to. Another disadvantage would be the impact of maternal antibodies in the first few months of infancy. At the same time since malaria is also related to occupation and population movements, it might not be sensitive enough to monitor such changes. Socially it might discourage all others to seek care knowing their blood slides would not be taken. The same argument would be applied to the 1-4 year olds except that the sample would be bigger and maternal antibodies would not have an impact. But recent evidence (Greenwood, 1991)

showed that changes in the 1 to 4 year olds would be more reliable than infants in malaria control. This comparative study raised another issue. The prevented fraction of malaria incidence by bednets over DDT was not as large as expected, as infants in the DDT group also slept under bednets, which itself was a good thing. The analysis needed to take proper considerations of how data were collected, in particular the sample was small when several interventions were compared.

Thus, based on the finding of the study, it is suggested that to adequately monitor and evaluate malaria control intervention in PHC, all that is needed is to take blood slides of patients under 10 years of age. This will include the more adventurous and active children, who, in the case of bednets, do wander outdoors away from bednets. This would also include all susceptible groups and the results would be sensitive to changes in intervention. At the same time it would reduce blood slides by an estimated 42% and thus save some considerable resources for other purposes. In public relations point of view, this would have more chance to be socially acceptable as it would include the primary school age group. This would satisfy and please the parents and would still provide adequate blood slides to occupy the microscopist. The data in the final analysis would pay special attention to the rate of P.falciparum in the under 10 year olds.

This suggestion would be well for the public and epidemiological purposes, but exceptions would need to be made for clinical use. The further suggestion in this regard would be to take blood slides of patients who were critically ill or unconscious to provide a diagnosis when malaria was a possibility. In such situations in peripheral health care the patient would be referred and a PCD blood slide taken and sent with the patient whose treatment would have been started. The third exception would be a patient who had received treatment but continued to be ill and drug resistance was suspected. In many cases such patient would also be referred after



the second line management (Schedule II, Annex D) failed. These three conditions would also provide useful epidemiological information in evaluating an intervention. The proportions with severe and complicated malaria and drug resistance (treatment failures).

These suggested changes though they appeared to be minor, would take much effort in re-training and re-orientation of health staff and health educating the public to co-operate. Therefore it would need active health training and education programmes. The suggested change has the prospect that resources would be saved, and much more meaningful and useful epidemiological information would be obtained. The changes as suggested would only need a modification of the established system in the malaria control programme, whereby PCD blood slides were taken only from patients under 10 years, which would provide an appropriate measure of malaria control in primary health care.

## **5.8 SUMMARY OF MAIN FINDINGS, CONCLUSIONS AND RECOMMENDATIONS**

### **5.8.1 The Main Findings and Conclusions**

The main findings in this multicentre DDT residual house spraying and permethrin impregnated bednets comparative trial were as follows;

- a. The endemicity of malaria in Solomon Islands was mesoendemic and was going through a transitional epidemiological change after years of low transmission. There were variations between and within regions and communities and the average API was between 15% and 40%.
- b. The infrastructure of a primary health care system that had participated in malaria control programme was in place as seen in many study communities with rural clinic nurses, Village Health Workers and PCD Volunteer Agents. This system was responsible for taking blood smears of patients suspected to be ill with malaria, and administer early



treatment.

- c. The main vector, A.farauti, completely avoided DDT sprayed surfaces, therefore was not killed even though it was still very sensitive with a 77% mortality by bioassay.
- d. The feeding of A.farauti was inhibited by permethrin impregnated bednets only by 47%. DDT did not inhibit feeding at all.
- e. Permethrin impregnated bednets reduced biting density by 57.8% and parity by 10%. The rates were lower indoor than outdoor. There was no impact by DDT on biting density, and mean parity which was averaging above 53%, was higher indoors than outdoors. A.farauti was highly sensitive to permethrin impregnated bednets, 100% mortality by bioassay, and a 77% mortality on those entering a shelter where a person slept under an impregnated bednet. A.punctulatus decreased so low that it became very rare with interventions especially permethrin impregnated bednets.
- f. The average sporozoite rate with A.farauti was 0.64%, higher rate indoor (0.99%) than outdoor (0.45%). The highest rate was indoor with DDT (1.42%) spraying and mosquitoes collected from the bednets area did not reveal any sporozoites, but the samples were small.
- g. Permethrin impregnated bednets reduced malaria prevalence significantly ( $p < 0.01$ ) more than DDT spraying. This was more marked in children and with P.falciparum. There were increases in the DDT group. The prevented fraction of malaria prevalence by bednets over DDT was 41.08%, which was increased in infants to 75% and particularly P.falciparum where an estimated fraction of 88% were prevented.
- h. Permethrin impregnated bednets decreased malaria incidence significantly more than DDT spraying ( $p < 0.001$ ). The difference were much larger in the young age group and with P.falciparum. The mean prevented fraction of incidence of malaria by permethrin

impregnated bednets over DDT was 64.47%. The fraction was highest in the 1-4 year olds, probably since infants in the DDT group were also sleeping under bednets, even untreated. About 65% of the incidence of infections that would have occurred as with DDT was prevented.

- i. The reductions with both incidence and prevalence with bednets decreased to about 10% and levelled out. It did not decline any further except in infants and younger children, whose rates also stabilised at some lower levels. There were little variations, confirming that permethrin impregnated bednets successfully contained transmission but was not effective enough to completely interrupt transmission.
- j. There was a high level of knowledge in the communities regarding the connection between malaria, mosquitoes and the interventions. The communities increasingly refused DDT spraying whilst compliance to bednets was continuously high.
- k. 60% of household members entered a house with treated bednets at 2000 hours and over 95% by 2200 hours. 20% slept under the bednets by 2000 hours and by 2200 hours this also increased to over 95%. It was the older children and adults, especially males, that remained outdoors contracting malaria outside bednets.
- l. Operationally, permethrin impregnated bednets cost an average of \$3.85 whilst DDT residual spraying \$8.53 per capita per year making permethrin treated bednets a cheaper alternative, including the annualized cost per bednet to be between \$2.64 and \$2.58 depending on the sizes.
- m. It took a minimum of \$0.40 to process and examine a PCD blood slide. The total blood slides examined in 1990 was estimated to have cost \$140,800.00, which was a lot of resources.



- n. It took a mean of 6.1 days for a blood slide to be made and examined, therefore it did not help with patient management. Only 20% of blood slides examined had any chance of benefitting patient treatment which took an average of three days. Therefore an estimated minimum investment of \$112,140.00 spent in 1990 on blood slides, just for epidemiological information, was an extravagant use of scarce resources in primary health care.
- o. The intervention that were studied had a major impact on young children, especially infants. Therefore ideally the most appropriate modified tool to be incorporated into a malaria control programme to measure effectiveness of an intervention in primary health care would be to set up a system to monitor only the PCD slides of infants. But for other technical and social reasons, it is suggested that a more practical system would be to take only blood slides of children under 10 years old, as an appropriate method to evaluate the effectiveness of malaria control in primary health care. This would save some resources which could be put to a better use at this level where resources are limited.

It is based on these findings that the principal conclusion that permethrin impregnated bednets, are not only more technically effective than DDT spraying in the study communities, but are a cost effective intervention in using limited resources in primary health care. The most appropriate evaluation system to measure the effectiveness of an intervention programme, which would also be cost effective, would be to modify the PCD mechanism in which only blood smears of patients under 10 years are taken.



### 5.8.2 THE RECOMMENDATIONS.

Therefore, the main recommendations to modify the malaria control policy and strategies in Solomon Islands are as follows.

- a. Permethrin impregnated bednets to replace DDT spraying as the principal vector control measure.
- b. Standardise impregnation procedures to enable communities to effectively participate in impregnating their own nets, every time they are impregnated.
- c. Only children under 10 years old, suspected of uncomplicated malaria to have their blood smears taken and examined routinely using the existing PCD network within primary health care. In addition patients with severe and complicated malaria, including those with cerebral signs and where drug resistance was suspected should have their blood slides taken. The final analysis, to evaluate and monitor the programme would especially be on the changes with P.falciparum in this age group.
- d. Re-training and re-orientation programmes to be carried out supported by simple but explicit instruction manuals on the recommended changes.
- e. Appropriate health education activities and strategies be strengthened and consolidated so that compliance on the use of bednets do not correspondingly decrease with the reduction of malaria, biting nuisance of mosquitoes and other insects.
- f. Continue strengthening and expansion of the PHC network with appropriate training, resources and other support, to maintain an adequate capacity to effectively undertake the recommended changes.
- g. Explore and set up effective ways for the community members to purchase bednets either individually or through setting up some community funds, and or subsidised by the malaria control programme with funds saved from no DDT and less blood slides. The

purchasing of the chemical remain as at present (as any changes would require modification of existing legislations).

- h. Finally, further appropriate studies to be done on the followings; determine the most appropriate complementary primary health care measure to impregnated bednets to solve local site-specific problems, continue monitoring insecticide sensitivity and explore the feasibility of alternative insecticides to permethrin, determine the effect of impregnated bednets on survival rate of A.farauti, and determine the residual effect of impregnated bednets after several re-impregnations of the same bednets which may lead to some cost saving measures.

If the programme is modified as recommended it would become much more a programme of malaria control in primary health care. Taking blood slides only as suggested would save resources to purchase other needed items as essential drugs, but still provide the needed sensitive information. Communities taking part in purchasing bednets and impregnation of bednets would create a sense of active participation that would lead to the situation in which the malaria control programme was for the community by the community.



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ORIGINAL THESIS

**ANNEX A STRUCTURED INTERVIEW/OBSERVATION QUESTIONNAIRE****BED NETS****Part One. General**

- 1 Date ..... 2. Village .....
3. Interviewer/Observer .....

.....

**Part Two. Interviewee**

- 1 Name ..... [ ]
2. Age ..... [ ]
3. Sex ..... [ ]
- 4 Position in Household ..... [ ]
5. Occupation ..... [ ]
6. Last level of Education..... [ ]

.....

**Part Three. Questionnaire****1. How do people get malaria?**

- a. Drinking dirty water/eating dirty food [ ]
- b. From dirty insects, eg. flies [ ]
- c. From mosquitoes [ ] [ ]
- d. Curse/devil [ ]
- e. Cold/rain? [ ]
- f. Do not know [ ]

**2. Do you own a mosquito net?**

- a. Own one [ ]
- b. Share one [ ] [ ]
- c. None [ ]

**3. List family members who own/share a mosquito net**

.....

.....

.....

**4. Do you use a mosquito net?**



- a. Yes ☐ ☐  
 b. No ☐

5. Why do use mosquito nets?

- a. Protect babies from insects ☐  
 b. Prevent mosquito bites ☐  
 c. Privacy ☐ ☐  
 d. Prevent malaria ☐  
 e. Others... Specify..

6. Why not use mosquito nets?

- a. Have no nets ☐  
 b. Nets torn and damaged ☐  
 c. Too hot ☐ ☐  
 d. Mosquitoes not a problem ☐  
 e. Others,... Specify....

7. Are you willing to buy mosquito nets?

- a. No, I have no money ☐  
 b. Yes, anytime ☐  
 c. Yes, only if cheaper/subsidized ☐ ☐  
 d. Other, ... Specify....

8. How often do you wash mosquito nets?

- a. Weekly ☐  
 b. Fortnightly ☐  
 c. Monthly ☐ ☐  
 d. Once a year ☐  
 e. Never ☐

9. Why do you wash mosquito nets?

- a. Smells ☐  
 b. Dirty ☐ ☐  
 c. Others,... Specify..

10. Any other comments?

.....

**Part Four. Observations**

1. The approximate time the house is visited. ... m/pm

## 2. Condition of nets

	1	2	3	4	5	6
No tears	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Less than 10 small tears	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
One or more large tears/more than 10 small tears	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

## 3. The approximate time the house is visited in evening, ....pm.

Name	Under Net	Inside house, outside net	Outside House
.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

DDDT

## Part One. General

1 Date..... 2. Village.....

3. Interviewer/Observer.....

.....

## Part Two. Interviewee

- 1 Name ..... ☐
2. Age ..... ☐
3. Sex ..... ☐
- 4 Position in Household ..... ☐
5. Occupation ..... ☐
6. Last level of Education..... ☐

.....

## Part Three. Questionnaire

1. How do people get malaria?

- a. Drinking dirty water/eating dirty food ☐
- b. From dirty insects, eg. flies ☐
- c. From mosquitoes ☐ ☐



- d. Curse/devil ☐
- e. Cold/rain? ☐
- f. Do not know ☐

2. Do you own a mosquito net?

- a. Own one ☐
- b. Share one ☐ [ ]
- c. None ☐

3. List family members who own/share a mosquito net

.....  
 .....  
 .....

4. Do you have your house sprayed with DDT during last spraying?

- a. Yes ☐
- b. No ☐ [ ]
- c. Partially ☐

5. If yes, why?

- a. Stops all insects ☐
- b. Stops malaria ☐ [ ]
- c. Health workers said so ☐
- d. Village leader said so ☐
- e. Others, Specify...

6. If no, why?

- a. Brings bed bugs ☐
- b. Does not kill mosquitoes ☐
- c. Kills pets ☐ [ ]
- d. Smells bad ☐
- e. Does not stop malaria ☐
- f. Do not like spraymen ☐
- g. Others, Specify....

7. Any other comments?

.....

### Part Four. Observation

1. Approximate time of day house is visited, .. am ..pm
2. The House visited is -

- a. Recently fully sprayed [ ]
- b. Partially sprayed (at least one wall unsprayed) [ ] [ ]
- c. Totally unsprayed [ ]

**3. The approximate time the house is visited in evening, ....pm.**

[illegible]



## **ANNEX B. MODIFIED MASS IMPREGNATION OF BED NETS**

### **"THE SOLO METHOD"**

#### **1. Materials needed.**

- a. 1 large plastic sheet measuring at least 3 m x 1.5 m
- b. 12 lengths of sticks
  - 4 - measuring 2.5 meters long and at least 60mm in diameter [A].
  - 4 - measuring 1.5 meters long and at least 40 mm in diameter [B].
  - 2 - measuring 2 meters long and at least 40 mm in diameter [C].
  - 2 - measuring 3 meters long and about 20mm in diameter [D].
- c. One coil (about 50 m) of nylon fishing line of size 120lbs test(at least).
- d. Several strong bush vines which villagers commonly use (may use nails if preferred).
- e. One 4 gallon plastic bucket (any container may be used).
- f. All normal impregnation equipment and chemicals.

#### **2. Steps**

- a. Fix the 4 large long posts [A] firmly into the ground at 4 corners forming a rectangular space, measuring 3m x 1.5m [diagram 1].
- b. Use the 4 shorter sticks [B] "choks", with one end fixed firmly to the ground and another tied firmly to the posts at 45° angle (diagram 2). This will absorb the tension by and on the lines.
- c. At least 1 m (or 1.5 m) above the ground level, firmly tie sticks [C] one on each end horizontally (diagram 2).
- d. On both sides tie the longer sticks [D] between the posts, just below the cross beams at both ends (diagram 2). These two sticks will be used to support the plastic sheet and also to absorb some tension at the top. (Bamboos may be used).
- e. Tie rows of nylon fishing line firmly across the beams at both ends (diagram 3). The lines are best 10cm or 15cm apart, and be of highest tension possible.
- f. Spread and suspend the plastic sheet below the lines by tying it to the bottom of the post across one end sloping down to the other end. At the other end the corners of both sheets are brought closer to make a 'gutter' for draining the insecticide into the bucket which is placed under it. The sides of the plastic sheet may be supported with strings tied to the smaller stick across the length (may be fixed with laundry pegs) (diagram 4).

### 3. Operation

Impregnate bed nets as usual but place the wet nets neatly in rows over the platform of nylon lines (diagram 5). The insecticide will drip onto the plastic and be drained back into the bucket for re-use. The nets may be turned around to drip. When they completely stop dripping they are hung up to dry.

When the nets are properly folded before impregnation, and arranged neatly on the platform by lines of nylon, the structure, with measurements as described, will support 40 to 60 nets at one time. Depending on the number of nets to be impregnated, more than one structure may be set up.

### 4. Advantages.

A single person may impregnate several nets at one time and within the shortest time necessary.

Saves insecticides for reuse.

Prevents irritating fatigue or backaches.

All materials needed (except plastic sheet and fishing line) are commonly available in the villages. Materials are easy to transport if required.

### Disadvantages.

Need to buy a plastic sheet and fishing line. (Plastic bucket should have been included in a set of impregnating equipments).

### 5. Notes.

Size of sticks depends on the strength of the wood used. Villagers always know. May use sawn timbers if available.

Villagers know the best bush strings, always ask.

Strong nylon string is preferred because of the tension required and also that absorption of insecticide by the lines from wet nets is kept to a minimum.

Best tip - Construct the system under a large tree for shade, or best under a suitable shelter if rain is expected, so that impregnation of several nets is not interrupted. The disturbance on the ground is easily levelled after the process.



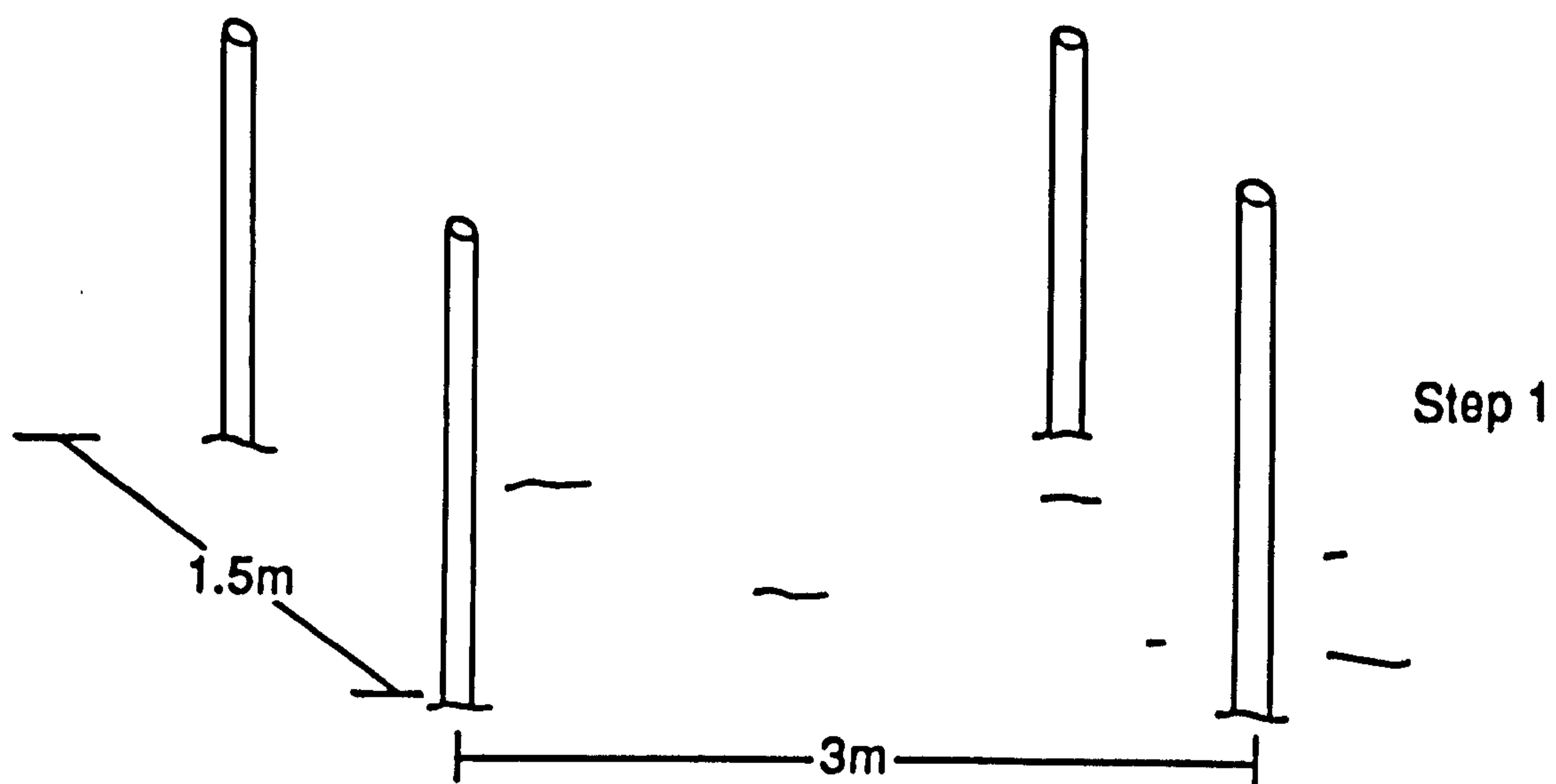
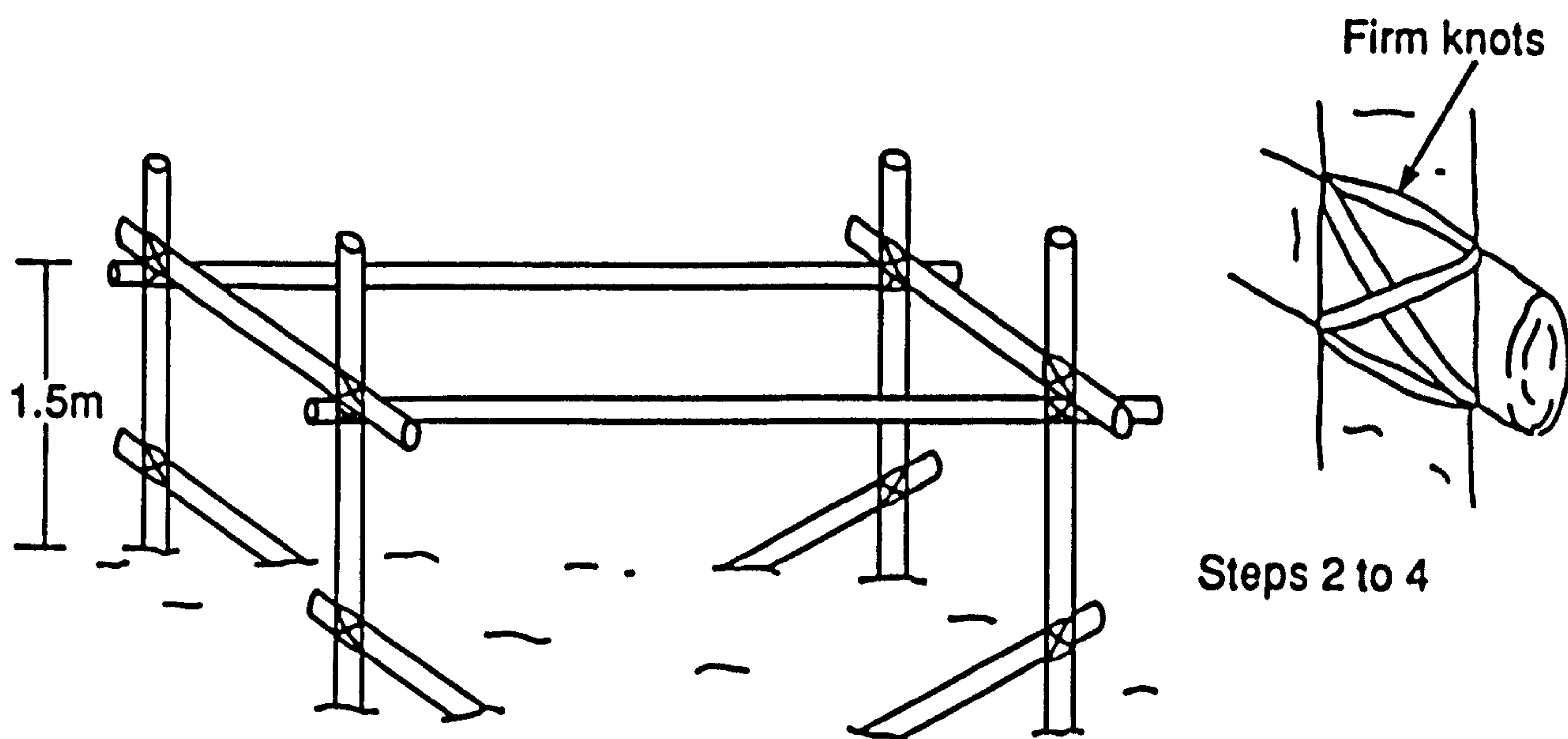
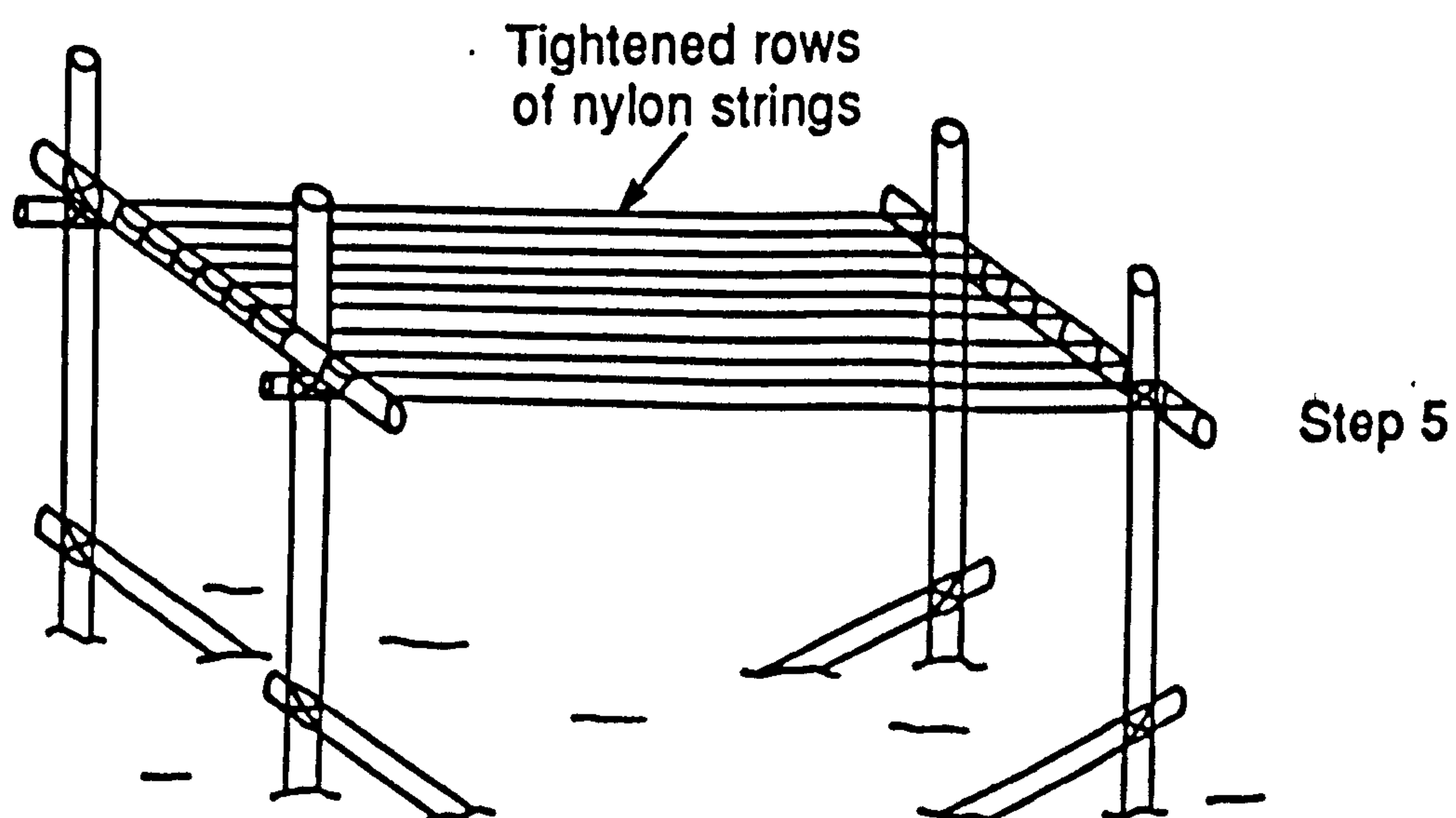


DIAGRAM A



Steps 2 to 4

DIAGRAM B



Step 5

DIAGRAM C

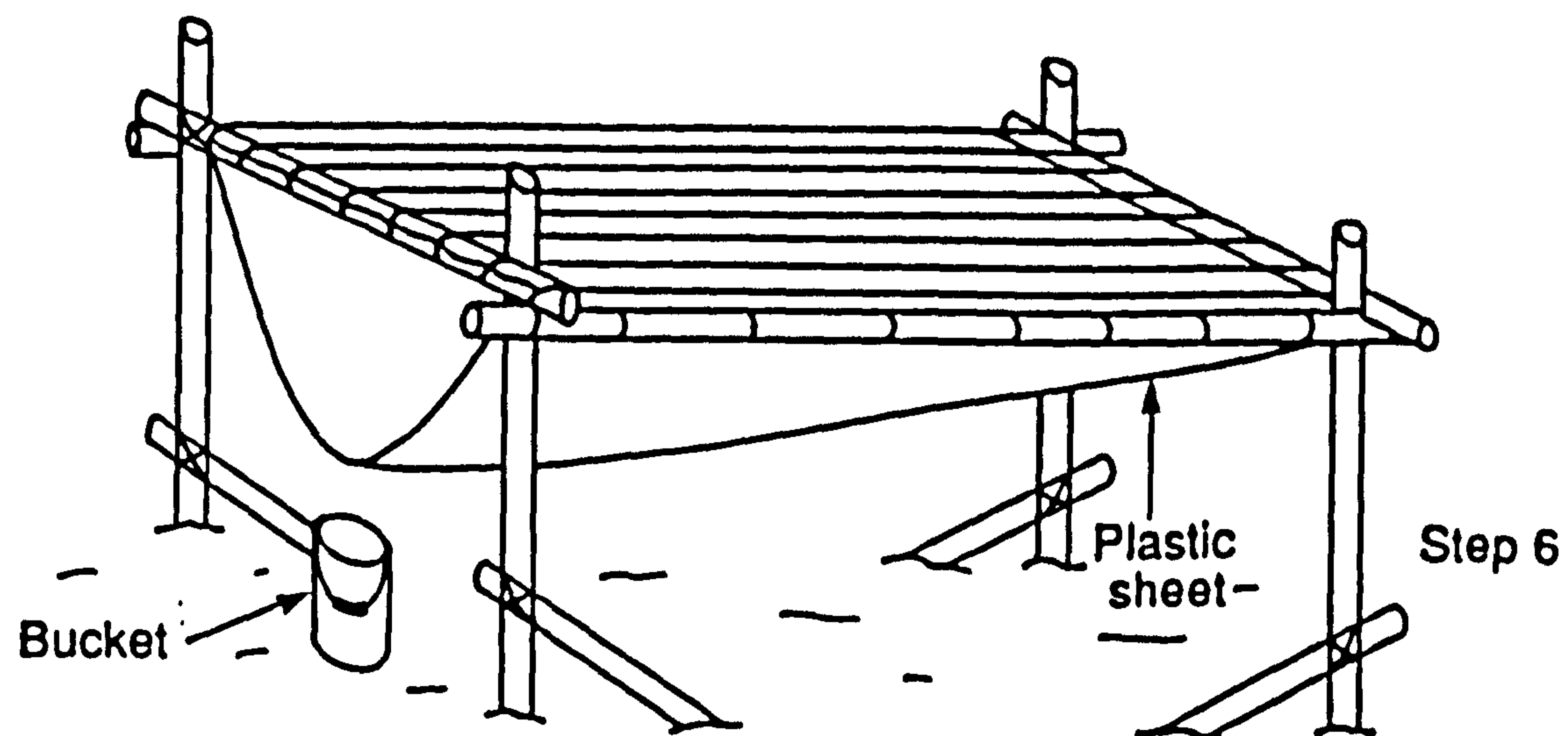


DIAGRAM D

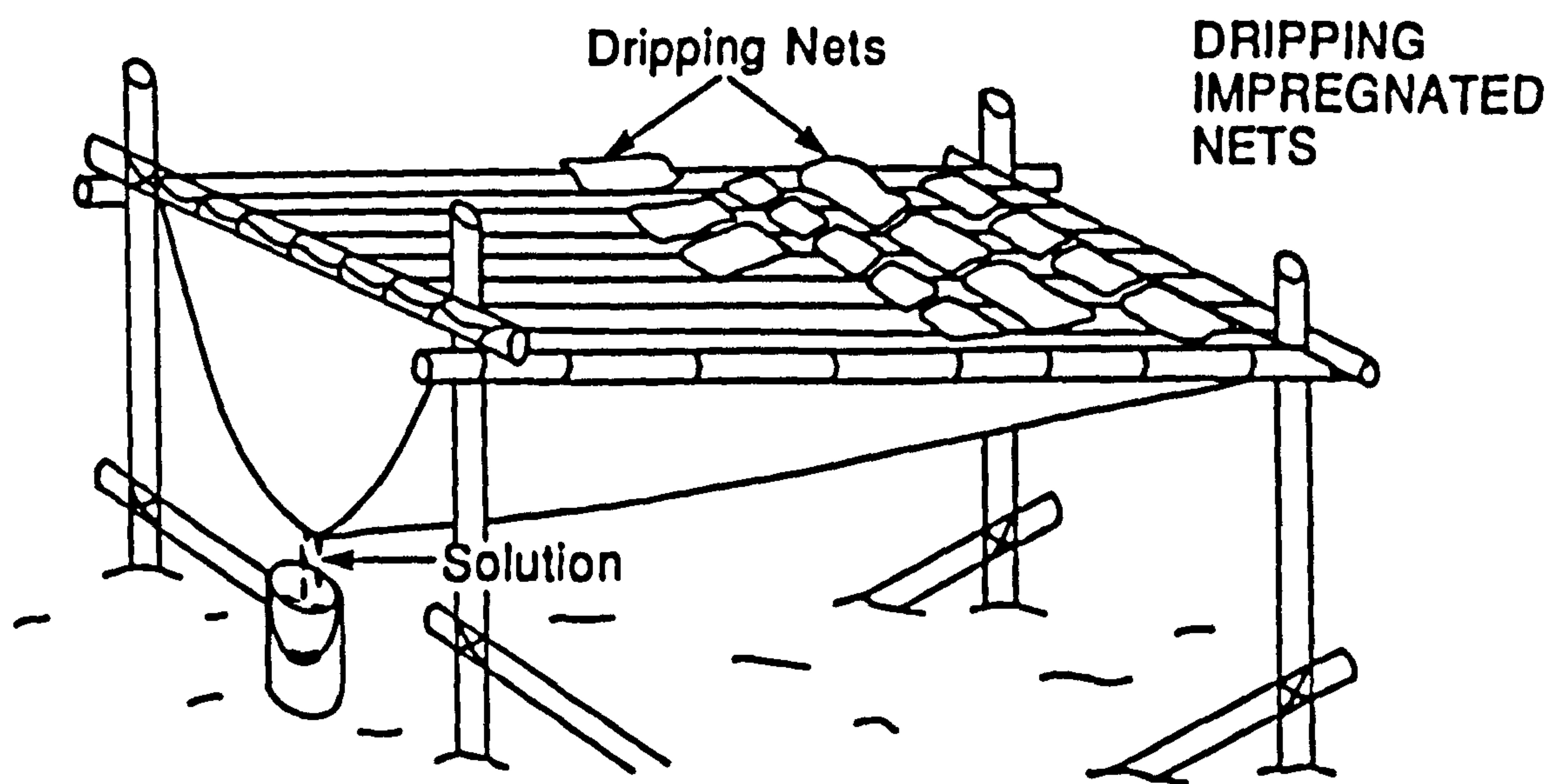
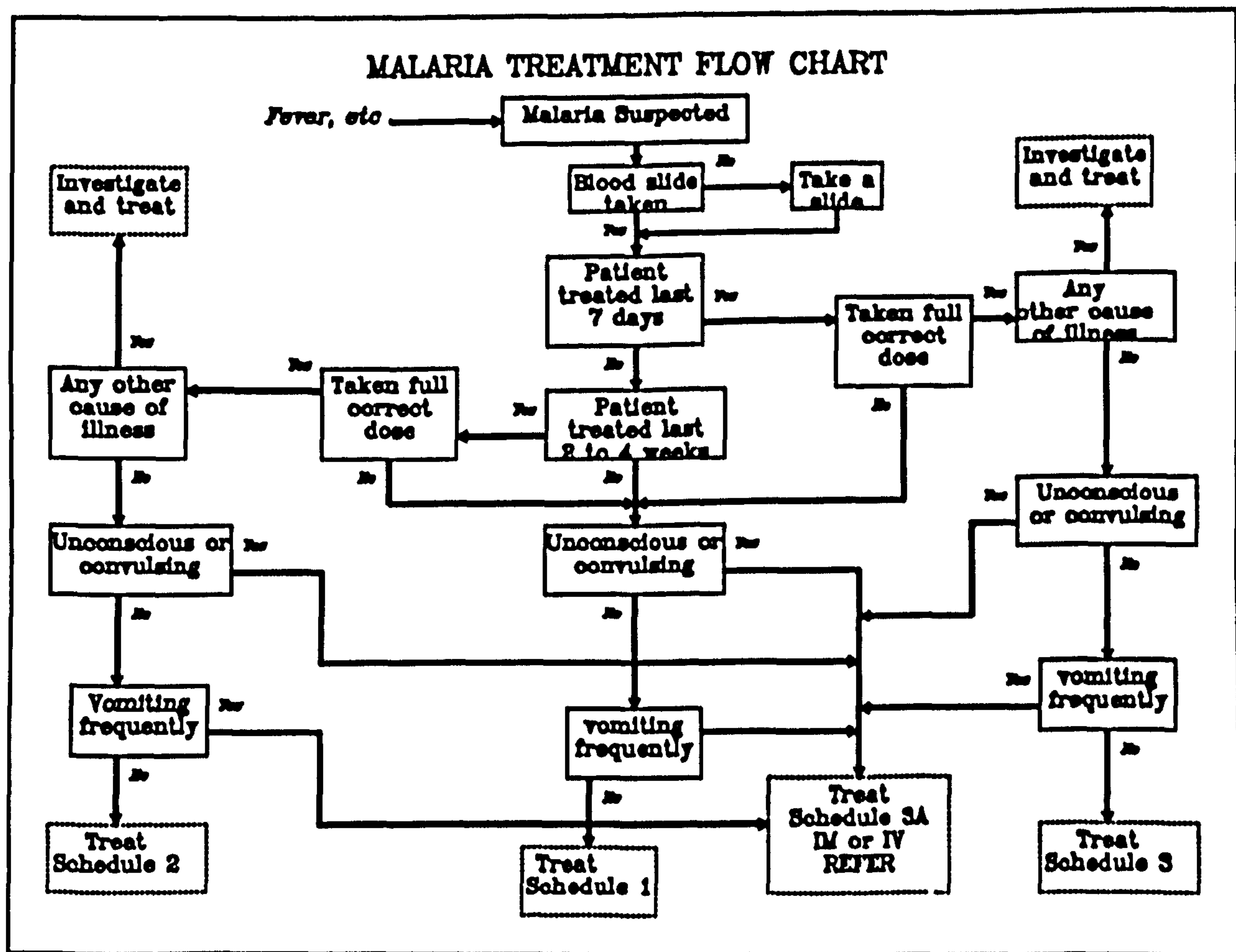


DIAGRAM E



# ANNEX C THE MALARIA MANAGEMENT FLOW CHART



## ANNEX D - THE MALARIA TREATMENT SCHEDULES

Schedule 1

### Treatment for Uncomplicated Malaria

DAY	DRUG	AGE GROUP				
		0-11month	1-4 yr	5-9 yr	10-14yr	15yr +
1	Chloroquine	$\frac{1}{2}$	1	2	<u>3</u>	4
	Primaquine	0	$1\frac{1}{2}$	3	4	6
2	Chloroquine	$\frac{1}{2}$	1	2	3	4
3	Chloroquine	$\frac{1}{2}$	1	2	3	4

Schedule 2

### Treatment for Grade I resistant *P. falciparum*

DAY	DRUG	AGE GROUP				
		0-11month	1-4 yr	5-9 yr	10-14yr	15yr +
1	Chloroquine	$\frac{1}{2}$	1	2	<u>3</u>	4
	Primaquine	0	$1\frac{1}{2}$	3	4	6
2	Chloroquine	$\frac{1}{2}$	1	2	3	4
3	Chloroquine	$\frac{1}{2}$	1	2	3	4
	Fansidar	0	1	$1\frac{1}{2}$	2	3

Schedule 3

### Treatment for Grades II and III resistant *P. falciparum*

DAY	DRUG	AGE GROUP				
		0-11month	1-4yr	5-9yr	10-14yr	15yr +
	Time	AM MD PM	AM MD PM	AM MD PM	AM MD PM	AM MD PM
1	Quinine 300mg	¼ ¼ ¼	½ ½ ½	1 1 1	1½ 1½ 1½	2 2 2
	Primaq.	0	1	3	4	0 0 6
2	Quinine	¼ ¼ ¼	½ ½ ½	1 1 1	1½ 1½ 1½	2 2 2
3	Quinine	¼ ¼ ¼	½ ½ ½	1 1 1	1½ 1½ 1½	2 2 2
	Fansidar	½	½	1½	2	3

Schedule 3A

### Intramuscular treatment for Severe and Complicated Malaria

Wt/kg			3	4-5	6-9	10-14	14-19	20-24	25-29	30-39	40-49	Adult
Day 1-3	Quinine 60mg/ml, twice daily	mg	30	40	60	120	150	210	240	300	420	600
		mls	0.5	0.75	1	2	2.5	3.5	4	5	7	10
Day 3	Fansidar		$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{2}$	1	1 $\frac{1}{2}$	1 $\frac{1}{2}$	2	2 $\frac{1}{2}$	3



ANNEX E. TABLES OF PREVALENCE SURVEY RESULTSTABLES OF RESULTS OF PREVALENCE SURVEYS IN ISABEL

Survey One

October 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	25	6	3	3	15	1	0	1
1-<5	86	28	14	14	80	15	10	5
5-<10	135	50	18	32	108	17	9	8
10-<15	120	37	17	20	123	19	8	11
15 plus	380	134	61	73	269	41	31	10
TOTAL	746	255	113	142	597	93	58	35

Survey Two

January 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Posst	P.f	P.v
<1	17	1	0	1	14	0	0	0
1-<5	86	8	5	3	76	9	7	2
5-<10	138	11	7	4	100	7	4	3
10-<15	118	9	5	4	130	16	5	11
15 plus	297	25	15	10	234	23	14	9
TOTAL	656	54	32	22	554	55	30	25

Survey Three

April 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	21	3	3	0	22	1	0	1
1-<5	110	18	11	7	77	7	0	7
5-<10	155	35	26	9	96	9	1	8
10-<15	118	23	11	12	119	17	1	16
15 plus	399	72	53	19	246	29	7	22
TOTAL	743	151	104	47	560	63	9	54

## Survey Four

July 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	21	3	3	0	25	1	0	1
1-<5	100	16	10	6	69	10	2	8
5-<10	136	39	28	21	73	14	6	8
10-<15	147	27	22	5	159	27	4	23
15 plus	358	65	46	19	245	30	10	20
TOTAL	762	150	109	51	571	82	22	60

## Survey Five

October 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	20	6	3	3	20	0	0	0
1-<5	92	24	18	6	63	5	0	5
5-<10	112	37	26	11	76	6	1	5
10-<15	181	39	21	18	132	24	5	19
15 plus	286	66	44	22	295	29	9	20
TOTAL	691	172	112	60	586	54	15	49

## Survey Six

February 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	17	1	1	0	21	1	0	1
1-<5	86	24	17	7	95	7	1	6
5-<10	148	48	37	11	126	10	3	7
10-<15	154	32	20	12	151	21	7	14
15 plus	302	93	78	15	294	50	20	30
TOTAL	707	198	153	45	687	89	31	58



## Survey Seven

April 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	17	6	3	3	15	0	0	0
1-<5	79	30	24	6	95	11	2	9
5-<10	91	28	14	14	101	16	7	9
10-<15	183	67	36	31	156	19	7	12
15 plus	315	94	40	54	285	37	19	18
TOTAL	685	225	117	108	652	97	35	48

## Survey Eight

July 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	23	7	5	2	23	0	0	0
1-<5	95	37	25	12	72	8	1	7
5-<10	99	56	33	23	92	10	2	8
10-<15	176	52	29	23	121	20	7	13
15 plus	316	80	51	29	298	24	5	19
TOTAL	709	232	143	89	606	62	15	47

TABLES OF PREVALENCE SURVEY RESULTS IN VELLA LA VELLA

## Survey One

October 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	21	5	5	0	22	4	3	1
1-<5	68	19	9	10	64	20	11	9
5-<10	75	16	10	6	87	15	8	7
10-<15	61	14	5	9	60	21	12	9
15plus	213	23	12	11	261	56	33	23
TOTAL	438	77	41	36	494	116	67	49

## Survey Two

February 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	22	4	2	2	17	1	1	0
1-<5	82	22	19	3	57	13	7	6
5-<10	61	15	6	9	78	12	6	6
10-<15	61	14	7	7	64	10	5	5
15plus	218	40	14	26	241	25	9	16
TOTAL	444	95	48	47	457	61	28	33

## Survey 3

April 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	18	6	6	0	13	0	0	0
1-<5	60	22	11	11	42	4	1	3
5-<10	51	17	11	6	64	8	3	5
10-<15	55	9	5	4	60	6	2	4
15plus	185	35	24	11	194	23	5	18
TOTAL	369	89	57	32	374	41	11	30

## Survey Four

July 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	20	5	4	1	11	0	0	0
1-<5	59	15	8	7	48	1	0	1
5-<10	76	21	15	6	63	6	1	5
10-<15	63	12	8	4	42	10	3	7
15plus	192	43	32	11	163	18	5	13
TOTAL	398	96	67	29	327	35	9	26



## Survey Five

October 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	19	3	1	2	14	0	0	0
1-<5	61	29	25	4	53	2	1	1
5-<10	72	17	12	5	59	4	0	4
10-<15	65	9	3	6	44	9	4	5
15plus	184	31	22	9	159	15	3	11
TOTAL	401	89	63	26	329	29	8	21

## Survey Six

February 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	17	5	2	3	10	0	0	0
1-<5	67	18	13	5	59	13	0	13
5-<10	78	11	9	2	54	8	1	7
10-<15	65	12	10	2	42	9	3	6
15plus	173	31	25	6	147	25	10	15
TOTAL	400	77	59	18	312	55	14	41

## Survey Seven

April 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	21	4	3	1	11	0	0	0
1-<5	68	23	19	4	53	1	0	1
5-<10	78	22	12	10	60	5	2	3
10-<15	64	17	9	8	49	10	4	6
15plus	193	36	33	3	142	15	5	10
TOTAL	424	102	76	26	315	31	11	20

Survey Eight

April 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	22	6	4	2	9	0	0	0
1-<5	60	30	21	9	49	3	0	3
5-<10	63	23	12	11	63	6	2	4
10-<15	58	15	9	6	52	7	2	5
15plus	185	23	13	10	163	12	4	8
TOTAL	388	97	59	38	336	28	8	20

**TABLES OF PREVALENCE SURVEYS IN VONA VONA/KOLOBANGARA**

Survey One

August 1989

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	21	2	2	0	4	0	0	0
1-<5	81	13	7	6	18	6	6	0
5-<10	108	16	8	8	22	8	7	1
10-<15	90	17	7	10	20	8	2	6
15 plus	253	31	17	14	46	10	6	4
TOTAL	553	79	41	38	110	32	21	11

Survey Two

November 1989

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	13	3	3	0	4	0	0	0
1-<5	76	11	8	2	17	5	5	0
5-<10	85	18	16	2	22	6	2	4
10-<15	79	18	14	4	35	8	4	4
15 plus	232	33	24	9	41	11	7	4
TOTAL	485	82	65	17	119	30	18	12



## Survey Three

February 1990

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	6	1	1	0	3	0	0	0
1 -<5	60	24	16	8	18	0	0	0
5 -<10	50	15	12	3	31	2	0	2
10-<15	56	7	4	3	29	2	2	0
15 plus	125	18	9	9	57	2	0	2
TOTAL	297	65	42	23	138	6	2	4

## Survey Four

May 1990

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	5	3	2	5	0	0	0
1 -<5	62	21	12	9	20	2	0	2
5 -<10	61	20	13	7	31	1	1	0
10-<15	51	15	11	4	29	4	2	2
15 plus	134	17	9	8	53	8	4	4
TOTAL	315	78	48	30	138	15	7	8

## Survey 5

August 1990

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	6	4	2	2	4	0	0	0
1 -<5	59	19	13	6	19	0	0	0
5 -<10	64	23	16	7	36	0	0	0
10-<15	65	11	6	5	19	4	1	2
15 plus	129	19	9	10	55	4	2	3
TOTAL	323	76	46	30	133	8	3	5

## Survey Six

November 1990

AGE Group	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	8	3	3	0	4	0	0	0
1-<5	73	33	28	5	16	0	0	0
5-<10	43	16	12	4	21	3	0	3
10-<15	54	10	5	5	33	7	4	3
15 plus	109	18	9	9	62	4	0	4
TOTAL	287	80	57	23	136	14	4	10

## Survey Seven

February 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	8	3	0	3	4	0	0	0
1-<5	62	27	16	11	21	0	1	0
5-<10	73	20	12	8	30	0	0	0
10-<15	72	12	8	4	31	5	2	3
15plus	136	19	10	9	59	8	2	6
Total	351	81	44	35	149	13	5	9

## Survey Eight

May 1991

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	3	2	1	5	0	0	0
1-<5	56	22	14	8	20	1	1	0
5-<10	68	16	11	5	33	3	0	3
10-<15	45	14	9	5	29	4	2	2
15 plus	121	21	14	7	60	6	3	3
TOTAL	297	76	50	26	147	14	6	8



TABLES OF PREVALENCE SURVEY RESULTS IN FLORIDA ISLANDS

Survey 1

December 1988

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	10	1	1	0	3	1	0	1
1-<5	36	20	8	12	19	4	2	2
5-<10	41	27	16	11	23	3	2	1
10-<15	45	16	9	7	19	5	3	2
15 plus	179	28	12	16	45	8	6	2
TOTAL	311	92	46	46	109	21	13	8

Survey Two

March 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	1	1	0	5	0	0	0
1-<5	26	14	5	9	21	6	3	3
5-<10	63	21	13	8	30	2	0	2
10-<15	45	8	6	2	25	3	2	1
15 plus	135	24	13	11	74	6	5	1
TOTAL	276	68	38	38	155	17	10	7

Survey Three

June 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	0	0	0	7	0	0	0
1-<5	27	8	1	7	21	3	0	3
5-<10	49	14	2	12	29	0	0	0
10-<15	24	6	4	2	24	2	1	1
15 plus	78	8	5	3	69	10	4	6
TOTAL	185	36	12	24	150	15	5	10

## Survey Four

September 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	5	2	1	1	4	0	0	0
1-<5	21	8	5	3	18	1	0	1
5-<10	37	11	8	3	29	4	1	3
10-<15	25	14	9	5	27	6	2	4
15 plus	83	25	13	12	72	10	4	6
TOTAL	171	60	36	24	150	17	7	14

## Survey Five

December 1989

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	6	1	0	1	5	0	0	0
1-<5	23	15	9	6	14	1	0	1
5-<10	39	24	18	6	21	4	2	2
10-<15	24	6	4	2	30	2	1	1
15 plus	105	29	21	8	55	5	3	2
TOTAL	197	75	52	23	125	12	6	6

## Survey Six

March 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	4	1	1	0	5	0	0	0
1-<5	21	10	6	4	15	3	1	2
5-<10	24	11	7	4	24	3	1	2
10-<15	26	11	5	6	21	6	3	3
15 plus	101	35	24	11	73	9	4	5
TOTAL	176	68	43	25	138	21	9	12



## Survey Seven

June 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	4	1	1	0	3	0	0	0
1-<5	34	17	9	8	21	4	1	3
5-<10	41	15	5	10	37	4	2	2
10-<15	24	15	8	7	13	7	3	4
15 plus	102	28	19	9	100	11	6	5
TOTAL	205	76	42	34	174	26	12	14

## Survey Eight

September 1990

AGE GROUP	DDT				BED NETS			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	1	1	0	4	0	0	0
1-<5	28	15	12	3	23	1	1	0
5-<10	35	12	8	4	27	4	2	2
10-<15	22	8	5	3	17	5	1	4
15 plus	99	30	22	8	92	10	3	7
TOTAL	191	66	48	18	163	20	7	13

TABLES OF PREVALENCE SURVEY RESULTS IN MALAITA

## Survey One

Sept.1989

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	1	0	9	0	2	2	0	2
1-4	12	9	9	0	11	9	6	3
5-9	15	10	8	2	22	15	8	7
10-14	12	8	8	0	14	10	4	6
15 plus	34	13	13	0	33	14	11	3
Total	74	40	38	2	82	50	29	21

## Survey Two

December 1987

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	1	0	0	0	1	0	0	0
1-4	27	14	6	8	16	6	0	6
5-9	28	9	7	2	19	8	3	5
10-14	15	4	2	2	17	7	5	2
15 plus	50	11	9	2	46	5	1	4
Total	121	38	24	14	89	28	9	19

## Survey Three

March 88

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	2	0	0	0	1	0	0	0
1-4	20	11	7	4	13	6	1	5
5-9	18	4	3	1	19	4	0	4
10-14	12	4	4	0	17	2	0	2
15 plus	18	2	2	0	32	6	0	6
Total	70	21	16	5	82	18	1	17

## Survey Four

June 1988

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	6	2	2	0	3	0	0	0
1-4	12	3	1	2	13	6	1	5
5-9	16	5	1	4	12	3	0	3
10-14	13	4	1	3	16	2	1	1
15 plus	31	2	1	1	36	3	1	2
Total	78	16	6	10	80	14	3	11



## Survey Five

September 1988

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	4	0	0	0	3	0	0	0
1-4	7	1	0	1	9	1	0	0
5-9	19	7	2	5	14	2	1	1
10-14	12	5	1	4	16	2	1	1
15 plus	43	6	4	2	28	2	0	1
Total	85	19	7	12	70	7	2	5

## Survey Six

December 1988

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	6	1	0	1	4	0	0	0
1-4	23	9	2	7	7	1	0	1
5-9	14	6	4	2	13	1	0	1
10-14	14	5	2	3	12	0	0	0
15 plus	37	3	1	2	30	1	0	1
Total	94	24	9	15	66	3	0	3

## Survey Seven

March 1989

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	3	0	0	0	7	0	0	0
1-4	14	3	3	0	15	1	0	1
5-9	13	9	1	8	21	2	0	2
10-14	16	5	2	3	14	1	1	0
15 plus	33	5	2	3	41	1	0	1
Total	79	22	8	14	98	5	1	4

## Survey Eight

June 1989

Age Group	DDT				Bed Net			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	3	0	0	0	3	0	0	0
1-4	22	15	15	0	15	2	0	2
5-9	23	17	13	4	26	4	1	3
10-14	18	6	6	0	14	2	1	1
15 plus	32	8	8	0	44	4	3	1
Total	98	46	42	4	102	12	5	6

TABLES OF PREVALENCE SURVEY RESULTS IN GUADALCANAL ONE TRIAL AREA

## Survey One

November 1988

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	1	0	0	0	10	3	3	0
1-4	12	6	2	4	29	13	11	2
5-9	12	4	3	1	60	28	24	4
10-14	10	3	2	1	28	4	3	1
15 plus	43	9	4	5	80	17	14	3
Total	78	22	11	11	207	65	55	10

## Survey Two

February 1989

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	0	0	0	0	8	1	0	1
1-4	33	13	7	6	36	15	12	3
5-9	44	9	7	2	42	17	15	2
10-14	37	8	5	3	21	9	7	2
15 plus	95	10	6	4	90	12	9	3
Total	212	40	25	15	197	54	43	11



## Survey Three

May 1989

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	3	0	0	0	6	1	0	1
1-4	26	11	8	3	35	8	2	6
5-9	18	3	2	1	50	24	9	14
10-14	15	1	1	0	33	10	2	7
15 plus	72	6	3	3	102	8	4	4
Total	134	21	14	7	226	51	17	32

## Survey Four

August 1989

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	0	0	0	0	15	0	0	0
1-4	15	6	4	2	38	8	2	6
5-9	11	6	3	3	81	13	6	7
10-14	5	1	1	0	38	9	6	3
15 plus	21	4	2	2	117	14	10	4
Total	52	17	10	7	279	44	24	20

## Survey Five

November 1989

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	7	2	0	2	14	4	0	4
1-4	63	23	18	5	51	16	7	9
5-9	81	32	13	19	38	9	4	5
10-14	65	26	9	17	19	4	3	1
15 plus	151	26	16	10	128	19	13	6
Total	367	109	56	53	250	52	27	25

## Survey Six

February 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	12	3	2	1	4	1	0	1
1-4	103	41	36	5	30	9	2	7
5-9	107	27	19	8	26	8	5	3
10-14	92	18	11	7	26	10	8	2
15 plus	183	36	23	13	83	21	10	11
Total	497	125	91	34	169	49	25	24

## Survey Seven

May 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	12	2	2	0	11	1	0	1
1-4	55	12	10	2	55	11	2	9
5-9	78	15	13	2	82	12	4	8
10-14	50	13	10	3	44	3	2	1
15 plus	127	17	14	3	147	18	12	6
Total	322	59	49	10	339	45	20	25

## Survey Eight

August 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	11	3	1	2	14	0	0	0
1-4	47	23	18	5	41	6	2	4
5-9	55	16	13	3	57	5	1	4
10-14	44	12	8	4	50	13	5	8
15 plus	126	20	14	6	91	4	3	1
Total	293	74	54	20	253	28	11	17



**TABLES OF PREVALENCE SURVEY RESULTS IN GUADALCANAL TWO AREA**

Survey One

December 1989

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	4	1	1	0	5	2	1	1
1-4	18	4	2	2	21	8	5	3
5-9	24	9	5	4	32	13	9	4
10-14	8	2	2	0	17	14	9	5
15 plus	45	16	9	7	72	29	12	17
Total	108	32	19	13	147	66	36	30

Survey Two

April 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	0	0	0	0	3	0	0	0
1-4	16	6	3	3	16	2	0	2
5-9	16	8	2	6	23	5	2	3
10-14	14	9	5	4	18	2	1	1
15 plus	43	4	2	2	52	13	6	7
Total	92	27	12	15	112	22	9	13

Survey Three

July 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	1	0	0	0	3	0	0	0
1-4	12	3	2	1	10	1	0	1
5-9	17	7	4	3	16	0	0	0
10-14	18	10	8	2	24	5	2	3
15 plus	42	16	8	8	71	13	6	7
Total	72	36	22	14	124	19	8	11

## Survey Four

October 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	3	0	0	0	2	0	0	0
1-4	12	4	3	1	10	0	0	0
5-9	16	5	2	3	15	3	1	2
10-14	12	2	0	2	26	5	1	4
15 plus	55	12	8	4	66	7	2	5
Total	98	23	13	10	119	15	4	9

## Survey Five

December 1990

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	2	0	0	0	4	0	0	0
1-4	11	3	3	0	20	1	1	0
5-9	23	9	7	2	19	3	0	3
10-14	16	8	5	3	16	3	1	2
15 plus	24	8	2	6	65	5	2	3
Total	76	38	17	11	124	12	4	8

## Survey Six

March 1991

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	1	0	0	0	1	0	0	0
1-4	13	4	3	1	24	2	0	2
5-9	25	13	11	2	19	2	0	2
10-14	17	7	2	5	18	5	1	4
15 plus	31	3	3	0	54	7	2	5
Total	87	27	19	8	116	16	3	13



## Survey Seven

June 1991

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	2	0	0	0	4	0	0	0
1-4	9	3	3	0	23	3	1	2
5-9	20	11	5	6	20	1	0	1
10-14	16	5	3	2	17	4	0	4
15 plus	39	4	1	3	58	4	2	2
Total	77	23	11	12	122	14	3	9

## Survey Eight

September 1991

Age Group	DDT				Bed Nets			
	Total	Pos	P.f	P.v	Total	Pos	P.f	P.v
<1	2	0	0	0	5	0	0	0
1-4	11	3	2	1	26	2	0	2
5-9	24	9	6	3	23	4	1	3
10-14	18	11	6	5	19	10	3	7
15 plus	52	12	3	9	59	3	2	1
Total	107	35	17	18	132	19	6	13

**ANNEX E. TABLES OF RESPONSES AND OBSERVATIONS IN SOCIAL SURVEY****Table 1: The level of formal education in the DDT and Bed nets groups.**

Intervention	Survey	Education Level (formal)				
		None	Primary	Secondary	Tertiary	Total
DDT	1	63	137	39	22	261
	2	43	97	13	6	159
	3	19	26	8	3	56
Bed Nets	1	99	215	33	18	365
	2	86	174	17	13	290
	3	47	81	9	4	141

**Table 2: The sex and age group distributions in the DDT and Bed nets groups.**

Intervention	Survey	Interviewee-sex		Interviewee - Age group		
		Male	Female	<20	20-49	50 plus
DDT	1	204	57	36	201	24
	2	136	23	63	83	13
	3	34	22	16	31	9
Bed Nets	1	263	102	101	221	43
	2	201	89	81	185	24
	3	98	43	13	116	12

**QUESTIONNAIRE RESULTS IN BED NETS****Table 3: What spread malaria, by number of households.**

SURVEYS	Survey 1	Survey 2	Survey 3
Eating/drinking dirty food	18	21	4
Mosquitoes	256	214	112
Devil/curse	30	20	6
Do not know	61	35	19
Total sample	365	290	141



**Table 4: Main reasons for using bed nets by number of households.**

SURVEYS	Survey 1	Survey 2	Survey 3
Prevents insects	15	59	29
Privacy	1	22	5
Keeps warm	2	30	13
Prevents malaria	12	158	82
Total sample	30	269	129

**Table 5: Main reasons for not using bed nets by number of households**

SURVEYS	Survey 1	Survey 2	Survey 3
Have no nets	259	17	8
Too hot	10	1	1
Mosquito, no problem	7	0	1
Nets torn	52	3	2
Total sample	328	21	12

**Table 6: Number of respondents willing to buy bed nets.**

SURVEYS	Survey 1	Survey 2	Survey 3
Yes, if cheap/subsidised	121	208	103
Yes, anytime	54	46	21
No	113	9	3
Not sure	76	23	14
Total sample	365	290	141

**Table 7: The frequency of washing bed nets.**

SURVEYS	Survey 1	Survey 2	Survey 3
Weekly	37	0	0
Fortnightly	95	15	5
Monthly	186	54	7
Annually	15	221	129
Never	32	0	0
<b>TOTAL SAMPLE</b>	<b>365</b>	<b>290</b>	<b>141</b>

**Table 8: Main reasons for washing bed nets.**

SURVEYS	Survey 1	Survey 2	Survey 3
Smells	216	67	46
Dirty	96	6	4
Others	21	217	91
<b>TOTAL SAMPLE</b>	<b>333</b>	<b>290</b>	<b>141</b>

**QUESTIONNAIRE/OBSERVATION RESULTS IN DDT GROUP**

**TABLE 9: What spread malaria by number of households**

SURVEYS	Survey 1	Survey 2	Survey 3
Eating/drinking dirty food	8	4	2
Mosquitoes	192	111	44
Devil/curse	27	12	2
Do not know	22	17	5
<b>TOTAL SAMPLE</b>	<b>261</b>	<b>159</b>	<b>56</b>

**Table 10: Number allowing houses to be sprayed with DDT**

SURVEYS	Survey 1	Survey 2	Survey 3
Yes	237	114	30
Partially	5	4	2
No	19	41	24
<b>TOTAL SAMPLE</b>	<b>261</b>	<b>159</b>	<b>56</b>



**Table 11: Main reasons for accepting DDT spraying.**

SURVEYS	Survey 1	Survey 2	Survey 3
Killed insects	58	20	5
Prevent malaria	154	63	19
Advised by health worker/village leader	29	34	12
TOTAL SAMPLE	242	118	36

**Table 12: Main reasons for refusing DDT spraying.**

SURVEYS	Survey 1	Survey 2	Survey 3
Brings bed bugs	6	11	4
Kills pets	5	10	7
Did not kill mosquitoes/ no stop malaria	4	15	11
Dislike spraymen	2	5	2
TOTAL SAMPLE	19	14	24